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EDITORIALS

THE TUMOR CLINIC IN THE GENERAL HOSPITAL

THE Tumor Clinic of the Memorial Hospital of Albany, N. Y. has been in existence for about three years. This is too short a period to warrant any attempt at a study of the end-results of the treatment of malignancy but is entirely adequate to illustrate the value and usefulness of such a clinic. In this country there have long been established clinics to care for patients with various maladies such as tuberculosis, venereal disease, diabetes mellitus, etc. but it is only of late that the idea of special clinics devoted to the treatment of malignant disease has received sufficient consideration. True, in a few of the larger centers there have been cancer hospitals which have done a great work but these institutions were not readily available for patients living at a distance. To overcome this difficulty, cancer clinics are being established in general hospitals and the cancer or tumor clinic is now a recognized development. Objections have been raised by governing boards of hospitals, both lay and professional, that establishment of such a clinic would lead to a gradual ingress of hopeless cases which would fill up hospital beds to the exclusion of other patients. Theoretically, this is a valid objection but from a practical point of view, it can easily be controlled to the satisfaction of all. Cancer occupies such a prominent place in the mortality tables, that the establishment of special clinics devoted to this problem is absolutely essential; such clinics must be established in locations readily accessible to patients; to many observers, the general hospitals in the communities to be served, seem the logical answer.

One of the great causes of the high mortality in cancer is delay in seeking competent medical advice. It is an acknowledged fact today that many early cases of cancer are curable. Therefore, if more cures are to be obtained, the public must be educated to seek medical advice early and the physicians must learn to recognize cancer in its early stages. The

establishment of a Tumor Clinic in a local hospital is very valuable in developing these two factors. Of necessity, some publicity follows the establishment of such a clinic. Meetings for the lay public are held at which nationally recognized authorities discuss cancer in simple language. As a result persons with abnormal swellings or tumors or unusual discharges or ulcers which do not heal, are stimulated to seek medical attention at an early date. Of course, objection has been raised that the public will develop "cancer phobia" which will tend to delay their seeking treatment but certainly such a "phobia" has not followed the publicity about tuberculosis and many other diseases. Perhaps this is the result of the old idea held by both lay and medical persons, that once a diagnosis of cancer was made, the fate of that individual was sealed. That such is not true is attested by the increasing number of so-called five-year cures in malignancy being reported before various organizations such as the American College of Surgeons. This organization considers the cancer problem of sufficient importance to devote an entire half day of the annual meeting to presentation of reports of five-year cures. Such facts must be presented to the public and the most satisfactory method is through the publicity of the Tumor Clinic. Knowing that such a clinic is established in a nearby hospital is of value to the patient directly. It makes him confident and desirous of securing modern diagnosis and treatment. He does not have to fear unconfirmed diagnoses or that treatment will follow the dictates of a single physician who may be partial to a single mode of treatment. To the community, the establishment of such a Tumor Clinic results in the saving of many lives, a lessening of suffering and lastly a real economic gain. Many persons are thereby restored to an earning capacity who otherwise would be a burden upon the charity of the community. Other individuals, who without adequate treatment would require continued hospitalization, continue to lead almost normal lives for long periods of time.

Of equal or perhaps greater importance, is the educational value of such a Tumor Clinic to the physicians of the community and the internes of the hospital. Primarily it makes them cancer concious so that they immediately consider cancer as a possible cause for unusual symptoms. It educates them to recognize promptly some of the signs of beginning cancer. Most of our previous cancer instruction has been based upon symptoms and signs occurring late in the course of the disease, at which time recognition means little. In the Tumor Clinic the physician is constantly seeing suspicious or early cancerous lesions in such numbers that a lasting impression is made. As a rule, the physician in general practice sees not more than ten cases of cancer a year, whereas in a clinic he will see at least ten times that many. The physician is taught by constant repetition the possible significance of the ulcerated cervix, the bleeding uterus, the small breast tumor. He sees and feels many of these; later he has opportunity of looking at sections of them and so fixes in his mind the entire picture.

A great deal of equipment is not required for the operation of a Tumor Clinic in a general hospital. A deep therapy machine of modern design is essential, for deep *x*-ray therapy is a very important part of the treatment of malignancy. A sufficient amount of radium, part of which is in needles of small radium content (1 to 2 mg.) must be available; for ordinary usages, not more than 100 mg. is needed. Frequently not all of this is in use, especially if the more modern technique of therapy is followed. In addition, a high frequency machine with satisfactory desiccating, coagulating and cutting currents is very useful.

Personnel seems to be the stumbling block to the formation of such a clinic in many general hospitals. Basicly, the Tumor Clinic must be built about the triad of pathologist, radiologist and surgeon. To these three are added qualified specialists

in the various branches of medicine and surgery. In our Clinic all the members are recruited from the hospital staff, selection being made on a basis of capability and interest in the cancer problem. All members of the hospital staff are urged to participate in the activities of the clinic and outside physicians are always welcome at any of the regular meetings. The tumor Clinic functions as a separate department of the hospital but is closely knit to many of the other departments. The hospital pathologist acts as head of our Tumor Clinic for he is not engaged in competitive practice; also he views the problem from the laboratory side rather than the clinical. The pathologist is probably the least partial member of the clinic. A registrar for the clinic is essential. We use a specially trained graduate nurse in that capacity; she also gives many of the deep therapy treatments under direction of the radiologist. In addition we have found a social worker to be a valuable asset in following up patients and in investigating their social status. We utilize one of the hospital workers for this purpose, on a part time basis which meets our present requirements.

The members of the Clinic meet weekly at a stated time. At these meetings they consider such cases as are presented for discussion of diagnosis or treatment. Also, at these meetings, the status of cases previously seen is reported and the patients seen if necessary. Whenever possible, the patients themselves are brought before the group. We have found this procedure stimulates and maintains interest on both sides. Cases with tumors requiring palpation are examined by two members only, in order to avoid possible metastases due to too much handling. The complete history, physical examination and laboratory data are presented with each new case; in reviewing an old case, merely an abstract is presented. If the clinic group is satisfied with the diagnosis, it recommends the treatment it thinks best for the individual. Thus each patient is individualized and one method of treatment is not used to the exclusion of other useful methods. When the diagnosis is not certain, steps to ascertain this are recommended before treatment is instituted. Obviously there arise instances where exceptions to this rule are made.

In all cases coming before the Clinic, it is a routine measure to have a blood Wassermann, complete blood examination and any special procedures as indicated. These tests are carried out in the regular hospital laboratory. Radiography is freely used to assist in determining the extent of the lesion and the existence of possible metastases.

Biopsy is done in most of our cases. To date we have not had any unfortunate experiences resulting from it. Fragments from superficial tumors are taken by scalpel or "radio knife"; pieces from more deeply situated tumors are removed by incision or aspiration with a large bore needle or the Hoffman punch. In some instances the entire tumor is excised for laboratory diagnosis. The biopsy gives us so much information as to the histology of the tumor and its sensitivity to radiation, that we feel justified in assuming the small theoretical risk of dissemination which may exist.

In all service or charity cases, the recommendations of our Clinic are mandatory and one or more members are assigned the duty of treating the patient. Those patients of this group requiring hospitalization are assigned to the physician or surgeon in the department indicated. Private patients referred to the Clinic for diagnosis and recommendations are returned to the care of the referring physician with such information. No treatment is given to the private patient except at the request of the referring physician. The latter is always requested to personally appear before the Clinic and present his patient for discussion; if he does not care to do so, the case is presented by the pathologist to avoid any semblance of unethical procedure. Perhaps this point of not giving treatment in private cases except upon request of the attending

physician is open to criticism. Usually the difficulty can be overcome by persuasion however and the plan invites cooperation rather than resistance.

In the treatment of all cases, the advice of the radiologist is freely sought and his cooperation is essential to the harmonious working of such a Clinic. As our Clinic has developed, some of the members have become more interested in certain limited fields of malignancy. The Clinic assists them in pursuing the studies by assigning to them for treatment the particular type of case they are interested in.

Finances of any clinic or hospital are always a worry. Our Clinic does a great proportion of free work but we have been able to meet all expenses incurred, by using fees paid for deep therapy and radium treatments. The entire equipment was purchased on notes which are being rapidly amortized. Private patients coming before the Clinic are charged a small registration fee but no charge of any kind is made in connection with charity patients. True such a department in a general hospital is not money making but if it can pay its own way, it more than justifies its existence from a financial point of view.

Briefly, the problem of treatment of some of the more common types of malignancy, as carried out in our Clinic, will be presented. Until a few years ago, the ethical physician had only two methods of attack upon malignancy, actual cauterization and surgery. The quack used caustic chemicals but this was considered an irregular procedure. A great many lives were spared for years by this combination of surgery and cautery. With the discovery of radium and a study of its use in the treatment of malignancy, a new weapon was added with resulting increase in the number of so-called cures. More lately, there has been added to the defense, deep *x*-ray therapy which has further improved the results. It is by various combinations of surgery, *x*-ray therapy, radium and electrosurgery that we have obtained the best results in our Clinic. Rarely is treatment by any single one of these modalities adequate so the physician or surgeon who favors one method to the exclusion of the others has no rightful employment in the tumor field.

In carcinoma of the breast, surgery is still the sheet anchor of treatment in the vast majority of cases. Nearly all of our cases, both public and private, are subjected to postoperative radiation. We approve of preoperative radiation also but find it difficult to persuade the patient, once operation is considered. Some cases of carcinoma of the breast, which we felt were inoperable, have been treated by deep therapy alone or in combination with radium with very excellent palliation. A few of these cases, thought to be in operable, have after radiation treatment become operable. Recurrences following operation, if few in number, have been treated by radium needles; with this method they seem to fairly melt away. Multiple recurrences have as a general rule been subjected to deep *x*-ray therapy as have extensions to the chest or more distant metastases.

In treating epidermoid carcinoma of the cervix uteri, we have come to feel that surgery has little place. We have depended entirely upon radium in and about the cervix, followed by deep therapy. Radium dosage has been large, usually between 5000 and 7000 milligram hours; the radium is filtered through platinum and rubber and the bladder shielded and also kept empty by a self-retaining catheter. Recently we have had a death due to peritonitis which followed insertion of radium into the cervix; apparently some infectious material from the ulcerated cervix was forced up into the corpus and out through the tubes by the plunger-like action of the radium capsule. This occurrence has made us reverse the order of therapy. We now give the patient a preliminary course of deep therapy and douches which eliminates much of the infection; then in about three weeks, radium is applied as before. Recurrences of the tumor are treated by deep therapy rather than by radium.

In dealing with adenocarcinoma of the uterine corpus, we place more confidence in surgery. After a preliminary course of deep therapy which in addition to inhibiting some of the tumor cells, minimizes any infection resulting from the curettage, a pan hysterectomy is done. In order to prevent any spill of tumor cells or infectious material from the tubes, these are ligated before operation is commenced. Recurrences are treated by deep therapy.

Carcinoma of the stomach still remains a surgical lesion and at present surgery offers the only hope of cure. Subtotal gastrectomy is the operation of choice and is performed even in the presence of demonstrable metastases unless these are too extensive. We feel that by this procedure, we are able to give the patient a great deal more comfort, while he lives, than can be given by a gastro-enterostomy. Recurrences are treated by deep *x*-ray therapy.

The same is largely true of the treatment of cancer of the intestinal tract. In cancer of the sigmoid and rectum we favor a combined abdomino-perineal operation, in one or two stages, depending upon the condition of the patient. As a rule preoperative radiation is given. A few of the cases, where the tumor lay low, have been treated by perineal excision and radiation. We have had but a very limited experience in treating rectal tumors by radium alone.

In the treatment of carcinoma of the bladder and prostate, deep *x*-ray therapy has been our first choice. In some tumors of the bladder, radon seeds have been planted, either through a cystotomy wound or through a cystoscope. Likewise in some cancers of the prostate, direct implantation of radium seeds has been done. However most of our success has been with deep therapy.

The treatment of tumors of the lymph glands, including Hodgkin's disease and lymphosarcoma, has been almost entirely by deep *x*-ray therapy. With such treatments, the masses rapidly disappear but only to reappear shortly in some other location. By keeping ever on the watch for new swellings and treating these promptly, we have been able to control quite a few of the cases of this group for a surprisingly long time.

Tumors of the skin, especially the epithelial ones, have been treated in the main by radium applications. In quite a few instances, where the tumor was of low grade malignancy, such as basal cell, excision or destruction of the tumor with the high frequency current has been followed by satisfactory results. In some instances where radium was used, a follow-up with deep therapy appeared to work very well.

Cancer of the lip has been treated by all methods with no great advantage apparent in any single method. Those patients who were subjected to surgical excision of the primary growth were subsequently given deep therapy treatments. Those treated by implantation of radium needles or by a moulage were subsequently given deep therapy treatments also. Our experience with block dissection of the glands draining the area is very limited and at present we prefer to attempt control of them by deep therapy rather than surgery.

In a paper of this type, which is intended merely to present general information about organization of a tumor clinic in the general hospital, further or more detailed reports do not belong. Such material will be presented at the proper time with attention to detail.

Arthur M. Dickinson.

EVALUATING THE RESULTS OF TOTAL THYROIDECTOMIES IN CARDIAC DISTURBANCES

RECENT radical operations on the thyroid gland have resulted in the demise of two very tenacious fetishes. *First*, it has compelled us to recognize that the basal metabolic rate does not tell all about what the thyroid gland is doing to the individual. *Second*, that there is no limit to the liberties the surgeon may take in the removal of the gland.

It is now generally recognized that the injury toxic thyroids exercise on the heart are not registered by the basal metabolic rate. It registers increased metabolism, nothing more, and as a diagnostic measure of the general mischief the gland may exert on the individual it is wholly misleading. That a partial removal of a goiter may cause astonishing improvement in cardiac function is generally recognized. The question as to the results on idiopathic cardiac disease by the removal of a normal thyroid gland compels the definition of a normal gland. Unfortunately this cannot be done. We can register what sort of a gland an individual supposedly normal possesses and that is all. That the normal gland possesses a cuboidal epithelium as the histologies tell us is untenable. Goldzieher has the courage to say without apology that it is flat. To refer to such epithelium as resting likewise is untenable because it lacks positive proof. Pathologists have devoted their entire attention to the structure of the epithelium. The histochemical changes that take place in the colloid have been almost entirely neglected.

To state that cardiac improvement follows total thyroidectomies in the absence of goiter is treading dangerous ground. Since the normal thyroid has no fixed size, whether or not the thyroid shall be regarded as enlarged must be a matter of personal opinion. We know that in Basedow's disease the tremendously toxic thyroid may show but little enlargement. Therefore the mere factor of the size of the thyroid gland is of but little use. If the slides show the thyroid to be normal, it means that the structure corresponds to the examiner's conception of what is normal. These glands in which cardiac improvement follows removal are characterized by a flat epithelium, very flat in fact for the most part. It should be remembered that what a slide shows cannot be accepted as a certain indication of the structure of the gland as a whole but only of that particular area. Other areas may show a more active epithelium. More commonly a more or less extensive basophilic colloid will be found with proper technique.

If there is pronounced improvement of the cardiac action after the removal of the thyroid gland it is well to renew the study. The gland must be thoroughly investigated in every part, not only the epithelium and colloid but evidences of degeneration. If this possibility is kept in mind one is forced to recognize the possibility that pronounced cardiac improvement is presumptive evidence that the thyroid gland was diseased and that the removal of it acted not so much in removing the whip as removing toxic products which were injuring the heart.

Furthermore the cardiac disturbances which the removal of the so-called normal thyroid helps are the same as those helped by the removal of old goiters in which all sorts of histologic changes can be demonstrated. It is all a difference of size of thyroid.

Nothing has been produced in an anatomic way to prove that an organic lesion of the heart is helped by the removal of a goiter, normal or diseased. That vascular lesions of the heart will regress is not to be expected. If symptoms of grave heart lesions disappear after complete thyroidectomies one must question the correctness of the diagnosis of organic disease.

The debatable thesis may therefore be stated as follows: When a heart lesion is helped by the total removal of the thyroid, the gland is not normal. One may use the effect the removal of the thyroid has on the heart as a measure to determine whether the thyroid was toxic or not.

ARTHUR E. HERTZLER

CYSTS OF THE OMENTUM

REVIEW AND REPORT OF A CASE

JOSEPH HORGAN, M.D.

Assistant Attending Surgeon, Childrens Hospital

WASHINGTON, D. C.

HISTORICAL DATA

IN 1852, W. T. Gairdner reported before the Pathological Society of London the finding at autopsy of "A Remarkable Cyst in the Omentum"; this was a simple serous cyst of the omentum and our first record of such. In 1881, Bantock brought to our attention a Hunterian specimen (No. 1109, Path. Series, Museum) of the Royal College of Surgeons, and since these discoveries reports have been made of other cases.

In 1910, Gueraine presented a thesis, reviewing the subject of cystic tumors of the omentum, and Dowd in 1911 presented a review of the literature, including 37 cases, to which he added one of his own. In his review Dowd attempted to classify the cases which had appeared in the literature up to that time. From his review he quite properly excluded the cysts of hydatid and malignant disease origin.

Since Dowd's contribution in 1911, there have been additional case reports which have added greatly to our knowledge of cysts of the omentum, giving further information as to their incidence, pathology, etiology, symptomatology, diagnosis, and treatment. Hence, we are now in a better position to classify these abnormalities but still cannot do it completely because of the meager information we have and the indifferent manner in which so many of the case reports on this subject have been presented. This fact caused Outerbridge (1914) to observe that: " . . . there are great differences of opinion with regard to the origin and significance of such cysts, and indeed comparatively few of the reported cases have been studied with sufficient care to permit of a definite determination of their point of origin and mode of formation."

It is the purpose of this paper to review the cases of cysts of the omentum, to bring the data to date, to report a case, and to suggest on the basis of these more complete data and from their apparent pathological character a classification of these cysts.

A thorough review of the literature has been made on this subject and will be epitomized in this paper. In doing this I have found acceptable 97 cases of cyst of the omentum, of which 48 per cent were classified as hematogenous and 52 per cent lymphogenous. The accepted cases are given in Table 1.

Pathologists' reports were available in 65 per cent of these case reports, and from them we find that the majority of these tumors were solitary (67 per cent), while decidedly less were multiple (33 per cent). In reviewing case reports care has been taken to include in Table 1 only those reports in which the diagnosis of cyst of the omentum was established by sufficient supporting data, such as operative observations, autopsy findings, or pathologist's reports.

All parts of the omentum are essentially the same in histologic structure. Cystic tumors have been found in all portions of it and represent a wide range of lesions. Those due to hydatid and malignant disease are most common. Kummer reported a case of cystic degeneration of an inflammatory process in the omentum which had been excited by a foreign body, a urethral catheter, which having been

TABLE 1
OMENTAL CYSTS

No.	Author	Date	Age	Sex	Single or Multiple	Type of Cyst	Result of Operation	Autopsy
1	Gairdner	1851	....	F	S	Hematogenous	.....	Yes
2	Simon	1858	44	M	S	Hematogenous	Died	
3	Piechaud	1877	42	M	S	Hematogenous	Died	
4	Pean	1880	40	F	M	Hematogenous	Recovered	
5	Ormsby	1883	26	F	S	Hematogenous	Recovered	
6	Rouiller	1884	76	M	S	Hematogenous	.....	Yes
7	Buckley	1885	34	F	S	Hematogenous		
8	Gallemaerts	1886	76	F	S	Hematogenous	Died	Yes
9	Gooding	1887	22	F	S	Lymphogenous	Recovered	
10	Nasse	1889	25	F	M	Lymphogenous	Recovered	
11	Wells	1890	4	F	M	Lymphogenous	Recovered	
12	Bellati	1891	22	F	S	Lymphogenous	Recovered	
13	Bellati	1891	48	F	M	Lymphogenous	Recovered	
14	Edebohls	1893	37	F	S	Hematogenous	Recovered	
15	Ris	1893	51	M	S	Hematogenous	Recovered	
16	Brandt	1893–4	15	F	S	Hematogenous	Recovered	
17	Krantz	1894	15	F	S	Lymphogenous		
18	Schwarzenberger	1894	5	F	S	Lymphogenous	Recovered	
19	Tilger	1895	71	F	S	Lymphogenous	Died	Yes
20	Erdheim	1896	52	F	S	Lymphogenous	Recovered	
21	Marfan	1896	2½	F	S	Hematogenous	Recovered	
22	Jessett	1896	....	F	S	Hematogenous	Recovered	
23	Hearn	1897	8	M	S	Hematogenous	Recovered	
24	Braithwaite	1898	4	F	S	Lymphogenous	Recovered	
25	Catterina	1899	45	M	S	Lymphogenous	Recovered	
26	Hoepfl	1901	3	..	S	Hematogenous	Recovered	
27	Jacobi	1901	7	F	S	Hematogenous	Recovered	
28	Marsh and Monsarrat	1901	1¾	F	M	Hematogenous	Recovered	
29	Sick	1902	21	M	S	Lymphogenous	Died	Yes
30	Schwartz	1902	43	F	S	Hematogenous	Recovered	
31	Boyd	1903	11	M	S	Hematogenous	Recovered	
32	Schramm	1903	1	F	M	Lymphogenous	Recovered	
33	Young	1905	9½	F	S	Hematogenous	Recovered	
34	Bidwell	1905	62	M	S	Hematogenous	Recovered	
35	Mathews	1905	8	M	S	Lymphogenous	Recovered	
36	Dewitzky and Morosow	1905	2	M	M	Lymphogenous	.....	Yes
37	Heinzelman	1906	30	F	S	Lymphogenous	Recovered	
38	Fort	1906	2½	F	S	Hematogenous	Recovered	
39	Wakefield	1907	4	M	M	Lymphogenous		
40	Karas	1907	38	F	M	Lymphogenous	Died	Yes
41	Giannettasio	1907	41	F	S	Hematogenous	Recovered	
42	Holleman	1908	19	F	S	Hematogenous	Recovered	
43	Costa	1908	....	M	S	Hematogenous		
44	Zembrzuski	1909	2	M	M	Lymphogenous	Recovered	
45	Himmelheber	1909	50	F	M	Lymphogenous	Recovered	
46	Seefisch	1909	4	M	S	Hematogenous	Recovered	
47	Rodman	1909	17	F	S	Lymphogenous	Recovered	Yes
48	Messa	1910	22	F	S	Hematogenous	Recovered	
49	Cordero	1910	53	F	M	Hematogenous	Died	
50	Betti	1910	19	F	S	Hematogenous	Recovered	
51	Gifford	1910	6	M	S	Hematogenous		
52	Dowd	1911	26	M	S	Lymphogenous	Recovered	
53	Stillman	1911	42	F	M	Lymphogenous		
54	Markoe (1)	1912	Adult	F	S	Hematogenous		
55	and (2)	1912	Adult	F	M	Lymphogenous	Recovered	
56	McPherson (3)	1912	27	F	S	Lymphogenous	Recovered	
57	Rochard	1912	23	M	S	Hematogenous	Recovered	
58	McLaren	1912	22	M	S	Hematogenous	Recovered	
59	Frank	1912	28	F	M	Lymphogenous	Recovered	
60	Guasoni	1913	5	F	M	Lymphogenous	Recovered	
61	Outerbridge	1914	34	F	M	Lymphogenous	Recovered	
62	Oliver	1914	65	F	M	Lymphogenous	Recovered	
63	Funk	1914	17	F	S	Lymphogenous	Recovered	
64	Ilott	1915	3	M	S	Lymphogenous	Recovered	
65	Laurenti	1916	70	M	S	Lymphogenous	.....	Yes
66	Kenney and Mason	1916	30	F	S	Hematogenous	Recovered	
67	Pybus	1917	4	F	S	Hematogenous	Recovered	
68	Borchers	1919	4½	M	M	Lymphogenous	Recovered	
69	Speese	1920	5	M	M	Lymphogenous	Died	
70	Duval	1920	27	M	S	Hematogenous	Recovered	
71	Vairakiam	1921	16	M	S	Lymphogenous	Recovered	
72	Oberlin	1921	27	M	S	Hematogenous	Recovered	
73	Oudard	1921	27	M	S	Hematogenous	Recovered	
74	Berti	1921	4½	F	S	Hematogenous	Recovered	
75	Arzela	1922	3	F	S	Lymphogenous	Recovered	
76	Copello	1923	31	F	M	Hematogenous	Recovered	
77	Chalier	1923	25	M	S	Hematogenous	Recovered	
78	Jerlov	1924	42	M	M	Lymphogenous	Recovered	
79	Jerlov	1924	30	F	M	Lymphogenous	Recovered	
80	Das	1924	12	M	S	Hematogenous	Recovered	
81	Rouffart	1924	35	F	S	Lymphogenous	Recovered	
82	Alberto and Guiseppe	1924	26	F	M	Lymphogenous	Recovered	
83	Lawrence	1925	3	F	S	Lymphogenous	Recovered	
84	Lay	1925	30	M	S	Hematogenous	Recovered	
85	Bernadet	1925	24	F	S	Lymphogenous	Recovered	
86	Kister	1925	4	F	M	Lymphogenous	Recovered	
87	Marinacci	1925	13	F	S	Hematogenous	Recovered	
88	Ryan	1926	4	M	M	Lymphogenous	Recovered	
89	Ladd	1926	8	F	S	Lymphogenous	Recovered	
90	Ladd	1926	8	F	S	Lymphogenous	Recovered	
91	Iselin	1927	Adult	M	S	Lymphogenous	Recovered	
92	Grausman and Jaffe	1928	44	M	M	Lymphogenous	Not stated	
93	Fisher	1928	4	M	M	Lymphogenous	Recovered	
94	Jackson	1928	50	M	M	Hematogenous		
95	Pariset	1932	64	M	M	Hematogenous	Recovered	
96	Peterson	1932	4	..	M	Hematogenous	Recovered	
97	Guillemot and Leveuf	1933	4	M	M	Lymphogenous	Recovered	

introduced into the uterus to induce abortion had found its way into the abdominal cavity. Dermoid cysts of the omentum have been recognized since they were observed and described by Ruysh in 1734 and Laflize in 1792. Whether dermoids arise as true omental cysts from rests of misplaced ovarian tissue is not apparent, but since dermoids originate most frequently in the ovaries it is probable that they are not all true omental cysts. Lazarus and Rosenthal (1931) observed a "synchronous dermoid cyst of the great omentum and of the ovary" and the pathological relationship of these cysts to the ovary has been observed by others. Ovarian cysts developing in the omentum with or without attachment to the ovary have been found by J. Greig Smith, Bantock and Doran, Firth, and Rose, but not under such conditions as would prove their origin in the omentum.

Cysts are frequently spoken of as tumors, but their nature as to structure and origin is different. By definition I have accepted the description by Adami, who states, "By the term 'cyst' we understand a sharply limited and abnormal accumulation of fluid in any area unprovided with a channel of outflow. The mere localized infiltration of a tissue with fluid, such as occurs in acute inflammation in local edema or elephantiasis, does not constitute a cyst; there must be a well-defined boundary wall or sac." Cysts may occur within tumors, single or multiple (cystomas), but have a structure that identifies them amongst other neoplastic tissue.

An attempt to classify these cysts of the omentum is not without its difficulties, as will be shown after an inspection of the incomplete data available. However, accepting the most apparent and probable mode of origin and formation, as suggested by the surgeon's reports and the characteristics apparent from the findings of the pathologists, it is possible to give a classification of these cysts as: (1) lymphogenous cysts; (2) hematogenous cysts.

The character of a cyst when not determined from its gross appearance, as for example in the cases of Karas, Schwarzenberger and Wakefield, was determined for classification with the aid of the pathologists' reports as "lymph cystomas" or "cystic lymphangioma." In Stillman's case the gross appearance of the cyst was that of a lymphangioma." In the cases of Guasoni, Jerlov (2 cases), Ladd (2 cases), Ilott, Borschers, Outerbridge, and Grausman and Jaffe, a diagnosis was made either by the author or the pathologist as "lymph cysts" or "cystic lymphangiomas." In the case report by Kister the omental cyst was described as a lymphatic cyst containing caseous material, suggesting the caseous degeneration of lymph tissue. Guillemot and Leveuf described a "cystic hemolymphangioma." I have classified this entire group as lymphogenous cysts.

Of those which seemed undoubtedly hemogenous there is one described by Oudard as a "sero-hematic cyst"; by Brandt, one which had "a thin, almost black fluid"; by Markoe and McPherson (Table 1, Case 54) one which had "coagulated blood and serum"; and by Simon, one containing "coagulated blood." In Edebohls' case the cyst contained "chocolate-colored, turbid fluid," and a similar observation was made in the cases reported by Giannettasio, Holleman, Costa, Pybus and Pariset.

Seefisch described the omental cyst he reported as containing "undiluted blood," while Chalier in his case spoke of "hemorrhagic fluid," and Ris described "a bloody pappy mass." Oberlin's and Betti's cases contained "red blood cells and cholesterine crystals"; Piechaud's case, "blood and albumen"; Lay's case, "a sero-purulent liquid"; and Das' case, "a dirty greenish fluid."

An interpretation of these variously descriptive expressions in reference to the conspicuous findings convinces one that many of them represent descriptions of the different stages of hemogenous cyst development as well as the changes which may be induced by infection, trauma, etc.

INCIDENCE

Omental cysts have been less frequently reported than all other cysts of the abdomen. This review of many cases, accepting but 97, suggests a ratio of 1:3 with mesenteric cysts, the next in rarity, as evident from the interesting paper of Peterson, who estimated that about 250 to 300 cases of mesenteric cyst had been reported up to 1932. It is interesting to note the relationship between the omental cysts and the mesenteric cysts as described by Peterson. He considered the two conditions similar in origin and histologic structure. This concept may be explained partially by their similar common origin embryonically and from the factors probable in their etiology.

In our series 35 per cent of the cases were found in the first decade of life and 68 per cent were found in the first thirty years of life. In this series the youngest patient was one year old, the oldest was seventy-six years, the average age was twenty years.

Race as a factor is not conspicuously associated with the incidence of these tumors but covers a wide range. There are no predominant racial tendencies.

The sex incidence is interesting, and its analysis indicated a majority for females, 60 per cent occurring in females, and only 40 per cent in males. This distribution in general is supported by the observations of Fort (1906), Hasbrouck (1908) and Geuraine (1910).

PATHOGENESIS

The pathogenesis of the simple cysts of the omentum presents a problem which remains unsolved. The dilatation of the lymphatics, degeneration of the lymph nodes and other lymphogenic factors, hemorrhage between the omental layers and other hemogenic factors, developmental errors, or embryonal rests, have all been suggested as etiological factors.

Young reported a case which suggests strongly the congenital origin of such cysts. Fort held the belief that in some cases they were of congenital origin, and other observers have expressed the same view, but no proof of such origin has been offered. Perhaps the most convincing evidence in this connection is the high incidence in childhood (35 per cent in the first decade of life).

Trauma as an exciting factor is emphasized by the histories given in the case reports of Piechaud, Gooding, Oberlin, and Chalier, but they represent only 4 per cent of the cases.

Ample evidence confirms the fact that escaped ovarian tissue has given rise to neoplastic tumors of the omentum. These, however, do not represent true primary cysts of the omentum. As an example of the manner by which they develop the case reported by Rose (1898) is of interest. He operated upon a woman and removed a multilocular ovarian cyst. At a later date she complained of a pain in the right kidney region, and a tumor mass, believed to be of renal origin, was found. When the patient was operated upon an incision was first made to remove what was thought to be a renal tumor. It proved to be another multilocular cyst of ovarian structure attached to the omentum. Phillips had a similar experience which he reported and stated, in reference to the second tumor removed, that he felt "the influence of the first laparotomy was such as to induce a very rapid increase in its rate of growth, an occurrence which has often been noticed by others; it seems likely that the increased vascularity has something to do with this, although some authorities attribute it to an alteration in the intra-abdominal pressure." Interesting though this is, it seems to disprove the omental origin of such tumors and I have not included them as primary cysts of the omentum.

Cystic degeneration in inflammatory processes may develop. Kummer, as already mentioned, described such a development about a foreign body, a urethral catheter which had been introduced into the uterus to induce abortion having found

its way into the abdominal cavity in contact with the omentum.

Hasbrouck advanced the theory that these cysts begin as an endothelioma between the fused surfaces of the omentum, which process presumably results from inflammation, and subsequently forms a closed sac. Contrary to this, Rokitansky believed them "of lymphatic origin and the result either of dilatation of the lymph veins or a cystic degeneration of the lymph nodes."

From the wide range of opinions one gathers that the majority believe these tumors to be of lymphatic origin, and this would appear justified from the data in the case reports. On the other hand, one becomes convinced of the hemogenic factor in the development of the lesser number of these tumors. Also, true hematomata of the omentum have been observed by Nixon, Krupp, and Green and Powers, the latter two observing "a large hematoma found in the gastro-hepatic omentum extending from the mid portion of the lesser curvature of the stomach upward to the cardia and outward for approximately 3 inches."

SYMPTOMATOLOGY

Omental cysts develop insidiously and the symptoms they produce are aggravated and produce increased clinical manifestations as the size of the tumor increases. The variability of the symptoms is affected by the complications which may be produced by pressure, torsion and traction. If the tumor is large, pressure on the adjacent viscera may cause gastro-enteric disturbances, such as nausea, vomiting after meals, anorexia, constipation or diarrhea. Pain, or other evidence of peritoneal irritation, dysuria, and frequent micturition are common symptoms.

Dowd found the complaint of pain in 86 per cent of the cases he reviewed. Pain and a sense of weight in the abdomen are increased by vigorous exercise and will subside with rest and while the patient remains at ease.

Emaciation and weakness were present in 36 per cent of the cases Dowd reviewed. Emaciation is sometimes a marked sign, as observed by Young (1905) in a patient from whom he drained 8 gallons of fluid from an omental cyst. Of the cases accepted in this review, 15 per cent were stated to be malnourished, 25 per cent were not malnourished and in 60 per cent the state of malnourishment was presumably not abnormal, since it was not mentioned. In the case seen by me there was emaciation and a secondary anemia, the hemoglobin being 66 per cent (Newcomer method).

DIAGNOSIS

These abnormalities of the omentum are of interest to the clinician because of the diagnostic problems which they present. To make a correct unqualified preoperative diagnosis of them does not seem possible with our present knowledge. Markoe and McPherson operated upon a patient for what was considered to be an inguinal hernia. Instead of a hernia it was found to be a prolapsed, pedunculated omental cyst. An "irreducible, inguinal hernia" proved at operation to be prolapsed omental cysts in the cases of Pariset, and of Alberto and Guisseppe.

Krupp in 1922 reported a case of omental cyst in which *x*-ray studies gave a picture markedly suggestive of carcinoma of the stomach, due, no doubt, to the pressure or traction of the cyst on the wall of the stomach.

Omental cysts have been most commonly confused with renal tumors. In the case which I shall describe a urologist was consulted. He thought it advisable to rule out by intravenous urography the presence of a hydronephrotic kidney. When this was done and an attempt made to rule out renal, hepatic, and gastrointestinal lesions, it seemed a choice between mesenteric and omental cysts, and was assumed to be a mesenteric cyst.

When the mass is regular in shape, freely movable, not tender to palpation, presenting no evidence of firm attachment

to the anterior abdominal wall or deep structures, it is reasonable to assume that it is cystic. But if the mass seems fixed, encroaches upon either flank, is high in the epigastrium, or in proximity to the pelvic borders, the problem is more complex. Certainly it is not feasible to make an unqualified diagnosis of omental or mesenteric cyst prior to operation.

TREATMENT

In the cases of cysts of great size, aspiration preoperatively of the fluid contents, with graded decompression of the intra-abdominal pressure, is advisable. Usually the removal of these tumor masses is simple, and they can be excised completely, care being taken to make an extracapsular dissection. When adhesions are diffuse, greater care in dissecting must be exercised. When necessary, fragmentary removal after aspiration is often possible, although less desirable than removal without rupture. When part of the cyst must be left, it is best that the edges of the raw surfaces be approximated. Except in very large tumors, or those undergoing degenerative change, it would seem rarely necessary to marsupialize the cyst.

When the condition of the patient and the size of the cyst preclude the surgical approach, aspiration combined with *x*-ray therapy may offer some advantage, as might be inferred from the excellent results obtained by Jerlov in the two cases so treated by him, one of which he reports recovered, the other improved.

REPORT OF CASE

An Italian boy, aged twelve years, was admitted to the surgical service of Dr. Edmund Horgan at the Children's Hospital on August 22, 1932 (Case #32-5257). His chief complaint was of a "lump in the abdomen." After study and preparation the patient was operated upon for the removal of an intra-abdominal cyst. He recovered and left the hospital twenty-five days after admission.

History: During the past year the patient had noticed a large lump in the abdomen at about the level of the umbilicus. This had increased slightly in size during the previous few months, the increase being most noticeable during the month preceding admission. There had been no constant pain, only a sense of discomfort when he ran, at which time he had a slight pain in the abdomen and a "fainty sensation." During the week prior to admission to the hospital pain and tenderness were felt by the patient when he pressed the mass.

The past history of infancy and childhood were clinically uneventful except for the usual childhood diseases. There was no history of operation, trauma, or other predisposing causes. The family history was of no clinical significance.

Physical Examination: The patient was an Italian boy apparently twelve years of age, slightly emaciated and malnourished, but free from pain and not acutely ill. The examination of the patient as he lay in bed revealed the tongue slightly coated, the teeth, although showing caries, were apparently in fair condition, and the tonsils were infected and hypertrophied. The heart and lungs seemed normal. The abdomen presented a large, apparently rather freely movable, smooth, cystic mass which, when moved about, returned to a position at the inner, inferior angle of the right upper quadrant. It was the size of a grapefruit, somewhat ovoid in shape, and not attached to the abdominal wall. It was not painful or tender to the touch. The spleen was not palpable, and the liver seemed normal in size and shape. Otherwise, the development was symmetrical and the structures normal.

Laboratory Tests: Repeated urinalyses gave normal findings. There was an anemia, secondary in nature, the red blood cells being 3,180,000 per cu. mm., and the hemoglobin 66 per cent (Newcomer method). The white blood cells gave a total count of 19,700 per cu. mm., of which 84 per cent were polymorphonuclear neutrophiles and 16 per cent lymphocytes. The polymorphonuclear cells were 76 per cent lobulated and 6 per cent bandform. The Wassermann and Kahn tests of the blood were negative. A routine stool examination was negative for occult blood, ova or parasites.

Diagnosis: With the foregoing information it was thought advisable to ask a urologist, Dr. Charles Howze, to see the patient in consultation. He suggested intravenous urography, and cystoscopy later if necessary, to rule out renal origin of the tumor. Roentgenologic

examination after the administration of the dye revealed the following: "There is a large mass in the right side of the abdomen. This is definitely outside the kidney. Intravenous pyelograms show the kidneys, pelvis and ureters to be normal."

A diagnosis of abdominal tumor, probably mesenteric cyst, was made, and the patient prepared for the surgical removal of the tumor.

Operation: The operation was performed under anesthesia induced with ethyl chloride and ether. Dr. E. B. Macon was the anesthetist. A paramedian rectus incision was made in the right upper quadrant to expose a large smooth, ovoid, cystic mass which was slightly blue in appearance. It occupied a place in the greater omentum, its long axis corresponding to the direction of the incision, with one pole in proximity to the greater curvature of the stomach. It was not adherent to the anterior abdominal wall. When the tumor was elevated, the transverse colon was found to be firmly adherent to its posterior surface, the tumor encroaching upon the omental bursa.

Three-fourths of the surface of the cyst was adherent, but it was possible to enucleate it in toto, leaving a large denuded area which was extremely vascular, but without edema. After removing the cyst the bleeding areas were ligated, and the bed was obliterated by continuous absorbable sutures. The wound was closed in layers, and supported with tension sutures.

The specimen removed at operation was examined by Dr. Clarence Rice, who reported his findings as follows: "Spec. No. 32–91–0. The gross specimen after fixation is an egg-shaped mass, 11 × 8 cm., weighing 475 gm. The surface is relatively smooth, with numbers of tags of tissue representing points of attachment to adjacent structures. Numerous blood vessels pass through the wall, giving a very adequate supply. When palpated, the tumor is relatively firm and gives the impression of enclosed fluid. The wall measures 1 mm. in thickness and is apparently of fibrous structure. On the interior, there are numerous collections of fibrin and red blood cells, giving

Fig. 1. Photomicrograph of omental cyst. Section through wall of cysts. × 50.

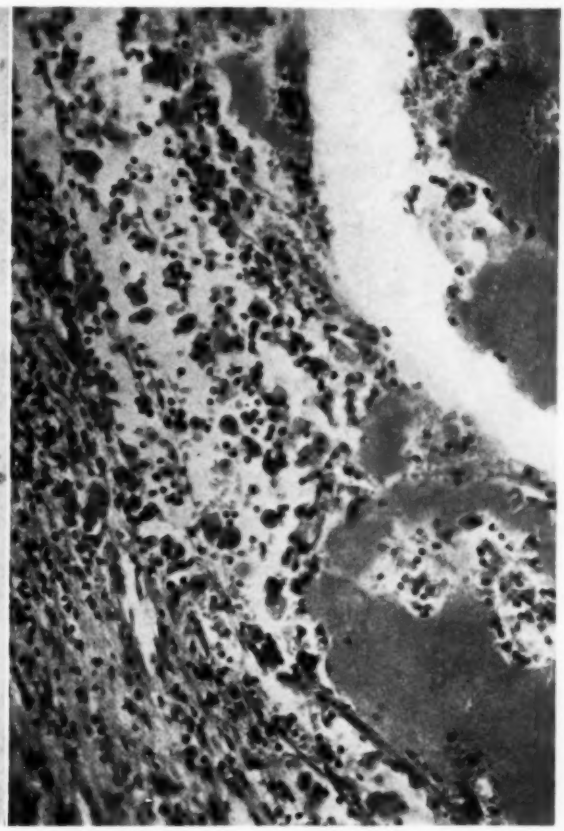

Fig. 2. Photomicrograph of omental cyst. Inner surface of wall of cyst. × 350.

a shaggy appearance. The cyst is monolocular. The wall encloses 250 c.c. of chocolate-colored, rather thick fluid. This is of high protein

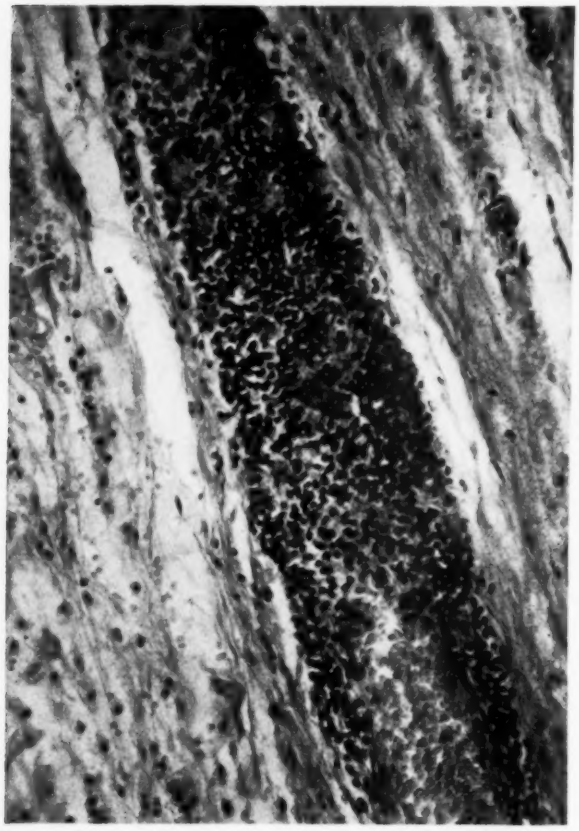

FIG. 3. Photomicrograph of omental cyst. Mid-portion of cyst wall showing collection of cells of lymph origin. × 350.

content, and when examined microscopically is found to be made up of red blood cells and finely granular material. No fat globules or crystals or other cellular elements are noted."

"On microscopic examination of sections of the specimen the exterior of the cyst is covered with endothelial cells, beneath which is a loose fibrous tissue. The wall is well vascularized and there is hemorrhage into the tissue (Fig. 1). Collections of round cells are found adjacent to some of the vessels. Infiltrating the wall and attached to the interior of the cyst cavity are numerous round cells and some polymorphonuclear leucocytes. Adherent to the interior of the cyst wall are collections of amorphous material with an admixture of erythrocytes (Fig. 2). Some areas show, in addition, collections of leucocytes and some strands of fibrous tissue (Fig. 3)."

"It is believed that the homogeneous material is of lymphoid origin and that the accumulations of lymph cells are developmental, not accidental or due to intercurrent infection. It would seem that this is a lymphatic

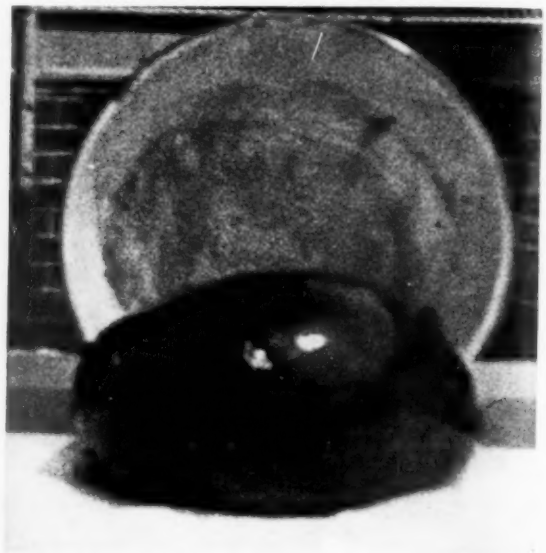

FIG. 4. Omental cyst removed by author from an Italian boy, aged twelve years.

cyst in which hemorrhage has taken place. Diagnosis: Omental cyst, not malignant."

Course: Aside from the development of a wound infection, which was drained on the fifth postoperative day, the patient recovered satisfactorily. His emaciation and malnourished condition did not favor rapid recovery, but with supportive care, the patient was discharged in good condition on the twenty-fifth postoperative day. His weight on discharge was only 52 pounds but had increased to 65 pounds when a check-up was made two months later.

SUMMARY AND CONCLUSIONS

Omental cysts are extremely rare. They are found free of adjacent structures in the abdominal cavity, developing in the potential space of the layers of the omentum, or they may be adherent to the abdominal parieties, or to one or another of the abdominal or pelvic viscera. They vary greatly in size, may be either single or multiple, occur most frequently in children and young women, and may first be noticed by the increasing size of the tumor, which in some cases may reach tremendous proportions. Complications may be present due to the size, relations and site of the mass, manifesting circulatory, functional

or traumatic effects upon adjacent structures, or excited by degenerative changes within the cyst.

They may be confused easily with other abdominal abnormalities and are difficult to diagnose. Differential diagnosis includes the elimination of ascites, ovarian cysts, tuberculous peritonitis, mesenteric cysts, gall-bladder disease, anomalies of the stomach, liver, or kidneys, pancreatic cysts, neoplastic and parasitic lesions, inguinal hernia, primary or metastatic malignant disease, etc.

The operative mortality, although indifferently reported in the literature appears to be about 12 per cent.

A likely origin of hematogenous omental cysts, as Hasbrouck has suggested, is from a fusion of the layers of the potential space of the omentum favoring the development of an endothelioma, which space later distends as spontaneous hemorrhage takes place within the potential space. Lymphogenous cysts are probably formed because of chronic irritation of a mild degree, with lymph exudation and cyst formation. Dermoid cysts have not been proven to have origin in the omentum but do develop from escaped ovarian tissue.

Aspiration, with preoperative decompression of the massive cysts, is indicated. Excision is the treatment of choice. When the adhesions do not allow a one-stage operation, exposure, fixation by suture to the anterior abdominal wall and drainage are necessary. As in all cysts the total excision of the sac is desirable.

The criteria for determining the etiology of these cysts are not known, but it is likely that they have either lymphogenous or hematogenous origin and that the majority are lymphogenous in character. In the case reported the tumor removed was apparently lymphogenous in character.

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RECENT DEVELOPMENTS IN COMBATTING

INFECTIONS OF THE URINARY TRACT*

ANSON L. CLARK, M.D.

OKLAHOMA CITY, OKLA.

UROLOGY, which has rapidly become one of the most highly developed branches of medicine, has undergone a most severe test in its attempt to combat infections of the urinary tract. As Young, Colston and Hill have emphasized:

> The difficulty of completely eradicating infections of the genito-urinary tract is due to the fact that the bacteria have usually penetrated so deeply that local treatment, whether of the urethra, bladder or kidney pelvis, usually fails to reach the depths of the infection. Although numerous new germicides have been produced, the problem still remains of how best to eradicate deep seated foci of infection. For many years internal therapeusis by oral administration of drugs has been used, sometimes effectively, but as a whole, this method has failed.

Should we require further proof that our treatment of this important group of infections is inadequate, we need only refer to the advertisements of the many new urinary antiseptics.

You will notice that I have refrained from mentioning the Neisserian infections in the title of my paper, although our inimitable Morrissey has frequently stated that the easiest way to obtain a large audience at a medical meeting is to announce the subject will be the "Treatment of Gonorrhea." One type of treatment applicable to gonorrheal infections which will undoubtedly be brought more forcibly to your attention in the near future is the use of fever therapy. My experience during the last six months with a number of patients has not as yet confirmed the optimistic results anticipated by the fever therapy enthusiasts in the treatment of acute specific infections in the male. However, in the treatment of both the acute and chronic Neisserian infections in the female, this type of treatment apparently has a distinct place, and may be extremely useful. Particularly is this so in preventing the extension of the inflammatory processes in the cervix and salpinx and in specific arthritis, which, as we all know, responds so poorly to the treatment in our present armamentarium. It is possible that the results in women have so far been more satisfactory because of the more active blood supply and because of the position of the organs in the female. Today fever therapy is undergoing many improvements, and with further refinements in the methods of application the way may be opened to a most useful adjunct in our treatment of this disease.

In the treatment of tuberculous infections of the urinary tract physicians have been able to do but little medically. Occasionally we are faced with the problem of attempting to ease the distress of the patient with a tuberculous ulcer of the bladder, where further help from surgical interference is no longer possible. In a few instances where the physician and the patient were both becoming desperate in an effort of one to help the other, the application of ultraviolet rays to the mucosa has had the desired results. In the female this may be accomplished with a slightly curved quartz applicator, using a water-cooled ultraviolet machine. In the male, a special quartz applicator used in the place of the obturator in the cystourethroscope, may be found the easiest means of applying this treatment.

* Read before American Urological Assn., New York Branch, Academy of Medicine.

Probably one of the most unsatisfactory bladder conditions to treat is the so-called elusive or Hunner ulcer. In a small number of such cases to which the ultraviolet ray treatment has been applied, considerable improvement has followed. Undoubtedly in the tuberculous bladder and the bladder where the Hunner ulcer is identified, the relief afforded by this therapy is only temporary; but both the patient and the physician involved are grateful for any measures which promise some relief.

Another problem with which the urologist has frequently to deal is that of infections of the urinary tract where the gram-negative bacillus is the offending organism. From among the many aspects presented by this subject I shall only emphasize those features which have particularly impressed me in my study of these patients, rather than reiterate much that has been expressed many times before.

This type of infection in children has long been believed to be ascending in origin, especially in girls. Although my opportunities to follow such cases in children have been limited, a most interesting series was recently accumulated by O'Conor, which I shall cite later. Helmholz believes the cause to be an upward extension of infection in nearly every instance.

Although in the past, focal infection throughout the body has been blamed as a source of the infected urinary tract, particular importance having been given to the tonsils, teeth and prostate, yet from my clinical observation I am led to believe that in the future the large intestine will be recognized as a prominent source of the infection. In women the cervix and ureteral glands should be carefully investigated.

The symptoms of a bacilluria are well known, but I should like to stress that I have seen many women who have been treated over long periods of time for symptoms resembling a chronic urethritis, relieved only when the concomitant mild bacilluria has been eliminated. In the male, also, where the outstanding complaints have pointed to a chronic posterior urethritis and prostatitis, the symptoms have disappeared when the growth of the offending bacillus has been inhibited.

Present-day management of infections of the urinary tract has recognized the importance of identifying the infecting organism. Wesson has recently complained that the advances in the refinement of bacteriology and laboratory technique make this process of identification far too difficult for the average clinician. Considerable experience has shown that, with the exception of the infrequent unusual case, the examination and diagnosis in the vast majority of patients is so simple as to require only a modest office laboratory equipment. The visual examination of the urine using the two glass test in the male and the catheterized specimen in the female, is the first step. The microscopic examination of the centrifuged sediment of the second glass in the male and the catheterized specimen in the female includes, first, an examination with the high-dry lens of the microscope and, second, the examination of the gram-stained preparation with the oil emersion lens. In office practice it has been my experience that culture of the urine is unnecessary except as confirmatory evidence or for making a prognosis as to the effects of treatment.

TREATMENT

In prescribing the ketogenic treatment for a patient, considerable time should be given at the initial visit to explain that a strict adherence to the rules is of prime importance. The adult patient is first instructed to take two ounces of castor oil on two successive mornings. A warm tap water enema is ordered each morning before the castor oil and at bed time. During these two days preceding the administration of the ketogenic diet, breakfast consists of fruit juices and coffee, while at the noon and evening meal only vegetable soup is allowed. The patient may have one or two bowls of vegetable soup, but starchy foods, such as crackers or bread, with the soup are prohibited. On

the morning of the third day following the two days of intensive elimination the patient commences the high-fat low-carbohydrate diet. In June, before the American Medical Association, Keltz and I presented a simplified dietary regimen which we have found to work very satisfactorily in office practice. Frequently, where patients are temporarily staying at a hotel or living under conditions where it is impossible to obtain a specially prepared menu, the extremely simplified food intake of one and one-half pints of cream and six eggs during each twenty-four hours helps in solving their problem. For desired results it is important to insist that extra thick cream containing at least 40 per cent butter fat be obtained, as the average whipping cream contains only about 32 per cent of butter fat. In addition the patient is allowed coffee or tea with which to dilute the cream, and butter, salt and pepper to make the eggs more palatable. At the same time he is cautioned against the use of any sugar or milk. The eggs may be cooked as desired and eaten at meal time with part of the cream, reserving the balance of the cream to be taken at mid-afternoon and at bed time. In some instances it has been most important to maintain a uniform ketosis throughout the twenty-four hour period. Examination has shown that, following a high-fat intake during the day, the ketone bodies may be present in considerable quantity during part of the night, while by morning they will be practically absent from the urine. It is important to avoid chewing gum, alkaline or sweet cathartics or any foods not prescribed.

The patient is asked to report the second day of the ketogenic diet. At this time a passed specimen of the urine is checked for the presence of diacetic acid and for the degree of acidity. The former test is made with equal parts of the urine and a 10 per cent solution of ferric chloride. With diacetic acid present the color of this mixture will vary from yellow to a port wine red, according to the amount of acid in the urine. In measuring the degree of acidity Herrold has suggested the addition of one drop of a .04 per cent solution of chlor-phenol red to 20 drops of the urine to be tested. At a pH of 5.2 or less there will be no evidence of a pink tinge. Above pH 5.2 the mixture of urine and chlorphenol red grade through various shades of pink to pH 6.4 where the color is definitely red.

After three or four days of the diet, if there is evidence of a satisfactory ketosis but the acidity of the urine is above pH 5.2, an attempt must be made to increase the acidity in order to fulfill the requirements for a satisfactory result. Any one of several drugs may be given orally to increase the acidity of the urine. Recently Crance has used nitrohydrochloric acid successfully. In the past I have used ammonium nitrate but following the appearance of mild nitrate poisoning in two instances, I have favored ammonium chloride. In a careful evalution of all the possible acidifying agents, Keith places ammonium chloride first, ammonium nitrate next with the acid solutions and sodium acid phosphate far below the first two mentioned. The selection of the drug to be used depends upon each individual case.

During the progress of the treatment a stain of the urine sediment is made every other day. When three such tests show that the bacillus is apparently no longer present in the urine, the treatment may be discontinued. Obviously the most conclusive final test is a urine free from bacilli after the patient has returned to a general mixed diet and the ketone bodies have disappeared from the urine. Where one can follow the progress of the patient conveniently, the microscopic examinations of the urine will eliminate the necessity for cultures of the urine as the final test as to whether the desired result has been secured.

During the course of treatment in women, all local medication is discontinued. In men, treatment is directed toward the prostate as a possible contributing focus. In this connection it must be recalled that

urethroscopic interference may be required where there are small chronic abscesses of the prostatic ducts. When the infection is of the mixed type a short course of neoarsphenamine, intravenously, may help to eliminate the coccic organisms from the urinary tract.

In prescribing the ketogenic treatment, to produce results the products of the changed metabolism must reach the infected part of the urinary tract. Poor renal function, particularly if unilateral, is a contraindication to this type of therapy. In carefully observed cases it was found that the largest part of the ketone bodies were excreted through the kidney which had the better function. In such an instance, if a poorly functioning kidney is infected, the inhibiting influence on the growth of the bacilli will not reach the infected area.

Statistical evidence of the success of any type of treatment is frequently of little value. Since an evaluation of the ketogenic treatment was begun three years ago, a considerable number of cases from various sources have been reported. It is safe to say that in approximately two out of three patients a successful result may be anticipated. In children the probability of eliminating the infection is even greater. A recent communication from O'Conor of Chicago giving the results of a most interesting investigation into the cause of recurrent infections in girls together with statistics as to the effectiveness of this type of treatment, follows:

About two years ago I was greatly upset over the fact that my second daughter, aged six, had had a bilateral colon bacillus pyelitis for one year and despite the fact that I had *not* treated her as the "shoe-maker's child" she was not particularly better than before I had commenced treatment. Her tonsils were cleanly out, no nasal or throat focus, normal bowel situation, repeated urograms showed no pelvic, ureteral or vesical neck obstruction. Yet she persistently wet the bed, was not eating well, and was mean and cranky with brother and sister, father and mother. This latter symptom has been particularly prominent in the story given me of similarly affected daughters by their parents. A cystoscopic treatment with bilateral ureteral catheterization and instillation of varying per cents of silver nitrate solution would bring about a marked improvement which would only last for six or eight days.

The problem of my own child got me interested in the ketogenic diet and I have utilized it as a therapeutic aid in fourteen girls between six and twelve years of age, all of whom have no demonstrated focal infection and no radiograph or catheter evidence of urinary stasis. In every instance ketosis was established and the urine cleared of pus and bacteria within five days to one month. All of these were carefully checked chemically, culturally, etc., so I won't go into unnecessary detail. *But* seven of these (50 per cent) would have gradual recurrence of pyuria, bacilluria and symptoms beginning as a rule the third week after returning to general diet. Needless to say, various forms of so-called urinary antiseptics were given all these little patients. When put back in ketosis they would again promptly clear up only to recur when again on regular diet. I then supplemented the acidulating and ketosis treatment with ureteral catheterization and silver nitrate instillation at intervals of three or four weeks. Three of the seven cleared up permanently, at least to date over six months in each instance. That left four that I had been seeing for over a year and a half that were not cured. One of these of course was my own child. I then went into a huddle with Clark and he went into a huddle with Helmholz and they suggested that I see if these four really had colon bacilli coming from the renal pelvis or just from the bladder. I smiled at this because I knew that originally they all had positive kidney cultures and I felt sure they still had or their bladder urines would not contain colon bacilli. *But* to my surprise on re-check I found that three of them had positive bladder cultures and negative kidney cultures. And one had only the left kidney positive. Then I got them in again and all four had positive colon and pus in the bladders and in no instance any pus or bacilli from the kidneys, and I got a lot of urine from each catheter and in very concentrated form. Was this then a persistent cystitis with pelvic lesions healed? Or was it an ascending infection from the urethra and at a stage when the

bladder was infected and the kidneys were not? At any rate back they went on the ketogenic diet, a rather foolish-sounding but necessary routine of external and personal hygiene was explained to the mothers and children, and where possible the children were brought in twice a week for bladder irrigation with weak silver solutions. Now three out of the four are able to go off the ketogenic diet, be careful about toilet hygiene, etc., and are absolutely cured symptomatically and with a half dozen or more negative urine cultures. The one who is not producing a sterile urine is so well that I have difficulty in keeping check on her urine. She is not my daughter thank goodness. I might add that these children were all in private practice and their parents were of an intelligent order.

I am sure you will all agree that O'Conor has aided us materially by his untiring effort and careful observation of these patients.

In two groups of cases there is apparently no explanation for unsatisfactory results. One group will not develop a ketonuria although the diet is carefully followed, while in the second group the acidity of the urine will not increase sufficiently even with large doses of some acidifying agent.

During the last year we have made an interesting study with the cooperation of six of the medical students at the University of Oklahoma Medical School who were placed on the ketogenic diet for varying periods of time. In brief the results were as follows:

The carbon dioxide combining power was reduced from an average figure of 60.1 volumes per cent before the diet was started to an average carbon dioxide capacity of 46.6 volumes per cent after five days on the ketogenic diet. This was apparently not a dangerous reduction in the carbon dioxide combining power. From numerous tests of the relation between the acidity of the urine and the concentration of the ketone bodies to bacterial growth, it was concluded that the effect of the ketogenic diet on infections of the urinary tract is bacteriostatic. It will be remembered that several years ago Fuller of London, in a careful investigation of ketourine, came to the conclusion that the beta-hydroxybutyric acid is the ketone body principally concerned in the bacteriostatic action. From our experiments we found that in order to secure sufficient bacteriostasis for satisfactory clinical results, a concentration of the beta-hydroxybutyric acid in the urine of .40 grams per cent must be obtained coincidental with an acidity of the urine of pH 5.2 or less.

CASE REPORTS

As briefly as possible I should like to review a few case histories which have been selected to illustrate essential points in the management of this type of therapy.

In the first example may I again ask you to note that for satisfactory results two factors are essential, namely ketone bodies in the urine and an acid urine of pH 5.2 or less.

CASE 1.

History: A man, aged fifty-four years, had during the previous eighteen months received treatment for burning on urination, frequency and urgency of urination, nocturia and perineal discomfort. Cystoscopic examination revealed a bilaterial pyelonephritis, chronic prostatitis and a verumontanitis. Because of the severity of his symptoms the patient had visited numerous medical centers where various types of treatment had given only temporary relief.

Examination: Macroscopically the urine was cloudy; microscopically the centrifuged second glass of urine contained pus iv; the stained sediment showed numerous gram-negative bacilli; the culture of the urine identified the organism as the Escherichia coli. The acidity of the urine was pH 6.3.

Treatment: Four days after the ketogenic diet was commenced the urine was clear; the acidity was pH 5.2. The urine sediment contained only a few gram-negative bacilli; the patient's symptoms were considerably improved. At the end of the eighth day of treatment the urine culture was negative; the acidity of the urine was pH 4.6; the symptoms had been relieved.

A letter from the patient two years later reported no recurrence.

Discussion: In this first case of bacilluria to which the ketogenic treatment was applied, it is of interest that the ketone bodies in the urine raised the acidity sufficiently to inhibit the growth of the bacillus. Occasionally during the first few days of treatment, the pH of the urine will decrease rapidly and then, as the body adjusts itself to the change in food intake, the pH will slowly increase. In such instances it is necessary to have the patient take ammonium chloride orally in order to increase the acidity of the urine. In adults where the urine is above pH 5.2 the results will usually be unsatisfactory.

During the period the patient is taking the diet it is essential that he make a daily visit to his physician. Many times there are questions which cannot be covered during the first instruction period and from my experience, no matter how emphatic the instructions or how carefully the cautions are printed, some deviation from the necessarily strict rules will occur. This was well illustrated by the following case.

Case II.

History: A man, aged thirty-nine, six months before, following a chill, had developed considerable frequency and burning on urination. His local physician had hospitalized him. After investigation of the upper urinary tract there had been recurrent systemic attacks with temperature as high as 105°F. He had remained in the hospital for six weeks. When first seen by us his complaints were recurrent attacks of chills and fever, pyuria with burning and frequency of urination. His wife added that there had been some question as to the possibility of a tuberculous condition of the urinary tract. He had lost a great deal of weight.

Examination: His general physical examination was negative except for badly infected tonsils. Both glasses of urine were cloudy and microscopically showed the presence of gram-negative bacilli. Intravenous urography revealed a moderate clubbing of the calices in the right kidney and some dilatation of that ureter. The catheterized specimen from both kidneys showed no pus but the colon bacillus was present in the culture of the specimens. The presence of any tuberculous infection was ruled out.

Treatment: When placed on ketogenic treatment the patient improved, but the diacetic acid did not appear in the urine in sufficient concentration. Upon careful questioning, the patient said that he was taking no food other than that prescribed, except a cup of coffee each evening. When asked what he was putting in the coffee, he admitted adding three teaspoons of sugar. As soon as this was stopped the ketone bodies in the urine increased and negative urine cultures were obtained within a week.

A report several months later showed his condition to be satisfactory.

Discussion: The three teaspoons of sugar daily which this patient was taking in addition to the planned diet, so upset the carbohydrate-fat ratio that ketosis was not obtained. Soda fountain drinks, fruits, chewing tobacco, chewing gum, sweet cathartics and milk of magnesia, if taken, will cause failures.

Undoubtedly the acidity of the urine in different individuals varies over a rather wide range. In the next case, while the acidity of the urine was high, some other bacteriostatic agent such as the ketone bodies was necessary to entirely eliminate the infecting organism from the urine.

Case III.

History: Mrs. W. J. F., aged forty-five, had sought relief from a chronic urinary tract infection for over ten years. Six years before the present visit a cystoscopic examination had shown a granular trigonitis, cystitis cystica and a negative upper urinary tract. At many different clinics almost every type of urinary antiseptic in vogue had been used, with frequent bladder lavage and instillations. Her complaints were recurring attacks of dysuria, frequency, ten to twelve times daily, with nocturia two to three times. One of the most distressing features to this patient was the interference with social activities because of the frequency of urination.

Examination: The catheterized urine was cloudy, with much epithelium, some pus and numerous gram-negative bacilli. The acidity of the urine at this time was pH 5.0. Cystoscopic examination showed rather severe

cystitis cystica confined to the trigone. The remainder of the bladder was apparently normal. Cultures of the urine from both kidneys showed no growth of organisms.

Treatment: The patient was placed on ketogenic treatment and ten days later the first negative culture was obtained. The distressing symptoms were entirely relieved. Four months after treatment was discontinued, a check of her condition showed negative urine with no return of symptoms. She complained now, that she was conspicuous socially because the frequency of urination had been reduced.

Discussion: At the time of our examination, before the dietary treatment was started, the urine was pH 5.0 but the colon bacilli were there in considerable number. During the ketogenic treatment, the pH varied from 4.8 to 5.3 with a steady decrease in the amount of infection until it was eliminated. This was of interest in demonstrating that ketone bodies in the urine were essential for satisfactory results.

When one is certain the patient is adhering strictly to the diet, and the daily test with 10 per cent ferric chloride shows that the ketone bodies in the urine have apparently reached a maximum and are diminishing, the diet should be discontinued. After a period on a general diet for ten days the ketogenic diet may be resumed effectively.

Case IV.

History: A woman, sixty-two years of age, came with the complaint that she had been voiding frequently and with considerable difficulty. At the time of the examination urination occurred every forty-five minutes both day and night, with most persistent burning at the bladder neck which was more severe when lying down and at the time of urination. As she was a school teacher this condition interfered with her work.

Examination: The catheterized specimen of the urine was very cloudy with pus grade IV and albumen grade I. Microscopically there were many gram negative bacilli which, on culture, were identified as colon bacilli. Cystoscopically there was a marked infection of the bladder and a careful search for a possible malignancy showed a small congested area in the posterior wall from which a specimen taken was reported as inflammatory tissue. Cultures of the urine from the upper urinary tract was negative.

Treatment: During the course of the ketogenic treatment it was noted that, after a rather strong diacetic acid test for several days, the test was showing a gradual decrease of ketone bodies in the urine. While the symptoms had improved considerably, the laboratory evidence still showed the presence of the infecting organism. At this point the patient was placed on a general mixed diet for ten days after which the ketogenic diet was repeated with a satisfactory result.

Discussion: Both the body and the infecting organism probably adjust themselves to the change instituted by the dietary treatment. When this is apparent, the diet will be more effective if preceded by a course of a week or ten days on a general diet. In this case, the first period of the treatment was given shortly after the biopsy of the bladder. Blood in the urine, of course, makes a better culture medium for the bacilli rendering it more difficult to retard their growth. When the bladder had entirely healed the second course of the treatment was successful.

Too frequently a patient is dismissed as neurotic when more careful diagnostic procedures might reveal the cause of the complaint.

Case V.

History: A woman, aged twenty-nine, had suffered for years with a chronic colitis. Eight months previous to our examination an acute attack of abdominal distress had been diagnosed as appendicitis. Two days later a severe urinary tract infection developed with the systemic reaction of chills and fever. Since then there had been recurrent periods of bladder distress with pyuria, frequency and burning on urination and nocturia three to four times. The types of treatment had been many and varied, both oral and local. At one time a daily bladder lavage had been given for four months with considerable relief. When treatment was discontinued the distress in the region of the bladder neck had returned.

Examination: A catheterized specimen of urine was clear, showed no albumin, no sugar and was microscopically negative for pus or red blood cells. Cystoscopic examination was negative except for a chronic granular urethritis grade 1. The stained smear of a centrifuged urine specimen revealed *no pus but numerous gram-negative bacilli*. Culture of the urine yielded Escherichia coli.

Treatment: Although this patient had been told that she had an intolerance for fats, when placed on the ketogenic treatment, the response was very satisfactory. It was necessary during the course of treatment to prescribe ammonium nitrate in rather large doses to acidify the urine sufficiently. During this period large numbers of casts were found in the urine. One authority explained that the diuresis produced washed the casts from the kidney tubules. When the infecting organism had been eliminated and the treatment discontinued the casts disappeared from the urine.

Discussion: Where there is a past history of urinary tract infection a careful microscopic examination of the stained urine sediment or a culture of the urine is important. In this instance the patient displayed considerable nervous tension. With the urine analysis and cystoscopic examination negative one might have placed the entire blame on the nervous system. When the real cause was located it was discovered that the nervousness was a result rather than the cause of her trouble.

Where the infection of the urinary tract does not respond satisfactorily to treatment the focus causing the recurrent bladder infection may be due to chronic abscesses of the prostatic ducts.

Case vi.

History: A man, aged fifty-nine, came with a complaint of burning on urination and urethral pain, of six months' duration. The pain was present constantly along the course of the urethra from the perineum to the external meatus. Moreover, there was some frequency of urination and nocturia one to two times. While there was no difficulty in starting the stream, no dribbling, no decrease in the size of the stream and no hematuria, the discomfort was so severe as to make it impossible for him to work.

Examination: The second glass of the two glass test showed pus grade II, albumin grade 1 with gram-negative bacilli in the microscopic examination of the stained sediment. Cystoscopically there proved to be a negative upper urinary tract, a mild cystitis and a marked prostatitis. In the prostatic urethra there were several dilated openings from which pus exuded during the cystoscopic examination. Several courses of the ketogenic treatment brought only temporary improvement. Between these periods prostatic massage and bladder irrigations were given. The prostatitis improved but little.

Under gas anesthesia the dilated ducts in the urethra were opened disclosing beneath one opening an old abscess cavity approximately 1.5 cm. in diameter. Following recovery from this procedure a course of the ketogenic treatment produced permanent satisfactory results.

Discussion: The removal of the contributing focus in the prostate by gentle prostatic massage or urethroscopic surgery is essential to successful treatment. Undoubtedly numerous bacillurias arise from prostatic infections. Conversely, when the prostatic infection responds poorly to treatment the cause may be an infection in the bladder or upper urinary tract.

The ketogenic treatment may be given though the patient has a history of having had attacks of cholecystitis.

Case vii.

History: Mrs. J. F., aged sixty, had had a diagnosis of cholecystitis twelve years before. This had improved on medical management. Within the last four years she had been receiving medication for some type of cardiac condition. During the last three months a severe urinary tract infection had developed. In addition to frequency of urination there was marked tenesmus. Urinary antiseptics had given some relief but the usual symptoms were present.

Examination: The catheterized specimen of the urine was cloudy with albumin grade II, pus grade IV, and red blood cells grade 1. Many gram-negative bacilli were present in the stained smear. The blood urea was within

normal limits although the combined intravenous phthalein test was slightly below normal. Cystoscopically there was a chronic granular urethritis, and chronic diffuse cystitis grade III. Cultures of catheterized specimens from both ureters showed the colon bacillus. The intravenous urogram was negative.

Treatment: After three days of the ketogenic diet there was considerable nausea and gastrointestinal discomfort. Water was given when possible, but all food was withheld for thirty-six hours. At this time the test for diacetic acid was strongly positive and the acidity of the urine was pH 5.3. Because of the age of the patient and her general condition it was felt best to discontinue treatment. Microscopically there was no evidence of the infecting organism, and the culture of the catheterized bladder urine was negative. Immediately after discontinuing the treatment, sodium bicarbonate and orange juice were given at alternate periods. After twenty-four hours a general diet was resumed.

Four months later this patient reported there had been no recurrence.

Discussion: An interesting observation in this and similar instances indicates that the patient who attains ketosis rapidly, with a rather marked reaction, eliminates the infection from the urinary tract in a short time. On the other hand when the patient apparently tolerates the diet and the ketone bodies appear in the urine slowly, a longer period of treatment is necessary.

The next example offers a most emphatic plea for control of the infecting organism, where this is possible, before cystoscopic interference is instituted.

Case VIII.

History: Mrs. T. M. B. gave a history of a pyelitis at pregnancy five years before. This had cleared up under treatment. Three months before coming under my observation she developed a severe urgency to urinate, with burning and frequency of urination. At that time a cystoscopic examination was advised by her local physician and a right retrograde pyelogram made. Following this manipulation she had a series of severe systemic reactions necessitating four weeks' hospitalization. The pyelogram showed a moderate right pyelectasis with dilatation of the right ureter grade I. At the time of my examination there was only moderate burning on urination, and no tenesmus.

Examination: A catheterized specimen of urine was cloudy and urine analysis showed pus grade IV and albumin grade I. A stained smear revealed innumerable gram-negative bacilli, with Escherichia coli in the culture of the urine. Instead of making cystoscopic or pyelographic studies immediately, this patient was placed on the ketogenic treatment and in eleven days the culture of the urine was negative. Cystoscopic examination at this time was negative. The intravenous urogram revealed a normal left kidney. The right kidney showed decreased function with a dilatation of the pelvis, calices, and upper half of the ureter grade II to III. With the symptoms relieved and the urine culture negative, the patient was advised to have no further treatment. Subsequent reports showed that the satisfactory results were permanent.

Discussion: It is most important in cases of acute or subacute bacillary infections to inhibit the growth and, if possible, eliminate the infecting organism from the urinary tract before extensive examinations are commenced. By an early retrograde pyelogram, little of value was learned at considerable cost to the patient. When the cultures of the urine were negative the patient was able to undergo a cystoscopic and pyelographic study of the urinary tract without reaction.

Following a prostatectomy or resection of the prostate gland more attention should be given to the infection so frequently found in cases of prostatic obstruction.

Case IX.

History: A man sixty-seven years of age presented himself with a history of a prostatectomy two years previously. Since that time his life had been centered around the resulting cystitis. Following instrumentation during one of his various courses of treatment he had developed a right epididymitis. At present his chief complaint was burning and frequency of urination with nocturia three to four times.

Examination: Urine analysis showed pus grade III with no albumin and no sugar. No

acid fast bacilli were found. Smear of the urine sediment showed numerous gram-negative bacilli and the report of the urine culture indicated the colon bacillus was the offending organism. The patient had a prostatitis grade IV. Intravenous urogram revealed no evidence of a lesion in the upper urinary tract.

Treatment: The patient was placed on the ketogenic diet. Every other day he received a bladder lavage with 1:2000 solution of acetic acid, followed by a gentle prostatic massage. Three weeks later after two short courses of the dietary treatment the urine culture was negative. He had no urinary symptoms, the urine was clear and he had a prostatitis grade I.

Discussion: Following an operative procedure on the urinary tract, the physician's duty to the patient is not ended. This is particularly important in cases where stones have been removed from the urinary tract, after which every effort should be made to eliminate the infection and thereby diminish the possibility of the formation of further calculi. Our experience has shown that immediately following prostatectomy or prostatic resection, the ketogenic diet is not indicated. It is possible the red blood cells in the urine accompanying the healing of the wound make a better culture medium for the organism. If it can be arranged, patients should report two months after the operation for check of their condition and at that time this type of treatment will be more effective. Having observed over 400 individuals who have received the ketogenic treatment for infections of the urinary tract it is apparent that this type of therapy, if carefully administered, is a useful addition to our therapeutic armamentarium.

DISCUSSION

Dr. Albert M. Crance (By invitation): The management of these infections seems to me to demand a complete urological examination before deciding upon any particular form of therapy.

Insofar as the action of the ketogenic diet is concerned and the manner in which it produces its bactericidal effect in destroying colon bacilli, we have had one theory from Clark early in his work to the effect that ketone bodies occurred in urines of patients upon the ketogenic diet. Later, Fuller of England wrote at length telling us that the specific occurring in the urines of patients on such a diet was an acid known as beta oxybutyric acid. I think we have sufficient evidence today from clinical experiences to agree as well as to disagree with Fuller.

Personally, I believe there are two factors which produce the curative effects when the ketogenic diet is used. First of all, we cannot have the presence of beta oxybutyric acid without the presence of diacetic acid and acetone. We have had several cases completely recover under ketogenic treatment that never did show any diacetic or acetone but their hydrogen ion concentrations went down below 5.1 and remained there for a few days. On the contrary we have had cases successfully treated by the ketogenic diet in whom the pH did not go below 5.4 but their diacetic and acetone tests became strongly evident. In other words, in the latter group the pH had nothing to do with producing sterile urines. These are the cases that fall under the theory of Fuller, and the colon bacilli were likely destroyed by the certain acid rather than any particular strength of acidity. In the first group with low pH and no beta oxybutyric acid present, it was the strength of acidity that did the work. Therefore we have two conclusions: It may either be a certain specific acid produced in these urines, or else it may be solely the strength of the acid which accomplishes the result.

In several papers I have repeatedly stated that colon bacillus infections in the urinary tract cannot in general be successfully managed without paying considerable attention to the etiology. It is very evident that intestinal stasis, or in fact intestinal upsets such as diarrhea, are frequently forerunners of urinary tract infections from this organism. If all such cases, many of which only have symptoms of cystitis, are studied carefully at cystoscopy, you will find that about 90 per cent of them show the origin of the infection to be in the kidney, one or both. This leaves but 10 per cent falling into the category of primary cystitis. I believe that the kidney becomes infected because of some intestinal irritation which crowds out colon bacilli into the lymphatics, and when they strike the kidney they become pyogenic or pus-producing organisms. It is just

as important, therefore, to treat the intestinal focus as it would be to remove abscessed teeth or infected tonsils when the predominating organism in a kidney infection happened to be a streptococcus. As a means of attacking this focus I have previously discussed a means of therapy termed the four-point treatment, simply because there are four parts to it:

1. Vaccine
2. Changing diet
3. Daily enemas
4. Acidophilus

In using this treatment it has been possible to obtain successful results in 80 per cent of the cases treated. This method works very well in the aerogenes type, and I still use it when failures are obtained by more modern methods. I am satisfied that the ketogenic diet or any other form of acid treatment will not attack the aerogenes type; and this brings us to the point as to what we mean by types of Bacillus coli.

For all clinical purposes we must consider this organism as occurring in two major types, the Aerobacter aerogenes and the Escherichia coli. A third one which may be considered is the Escherichia communior, which for all practical purposes may be included under the aerogenes group because its characteristics are very similar. It is true that in the Bergey Manual of Bacteriological Classifications there are some twenty-two varieties of the escherichia described, and seven others besides the aerogenes under that group. It would be most confusing to regard this fact as having any clinical significance, but I do believe that we should never be satisfied with a bacteriological report to the effect that colon bacillus is present. We must go further than this and know with which type we are dealing. The laboratories must use the fermentation test. The simplest way to distinguish one from the other is to subculture direct from the agar slant into a U tube containing sacchrose. The aerogenes type will produce gas within forty-eight hours, whereas the Escherichia coli type will not. Both types ferment and produce gas in lactose.

If one cares to use the ketogenic diet, he should restrict it to the Escherichia coli type. Many unnecessary failures with Dr. Clark's method of treatment have been brought about because all types were being treated in the same manner, perhaps unknowingly. If I were to criticize anything that Dr. Clark has said, it would be the manner in which he, as well as Dr. Helmholz, has persisted in using the ketogenic diet in the aerogenes type. My failures with this diet in this type of case have been 100 per cent.

A few weeks ago, at our Western New York and Ontario Branch meeting held in Toronto, Dr. Thomas W. Maloney and I gave a preliminary report on the use of a new acid method with which we are able to obtain rather good results through the use of nitrohydrochloric acid in full strength, diluting it in water so that the adult patient gets 10 minims of acid four times daily. We have been able with this treatment to obtain results in some cases much quicker than with the ketogenic diet. In a general diet with the use of this acid we have been able to obtain a pH as low as 4.9 in less than one week. Because of the terrific expense in times such as these we all know that the average patient cannot afford hospitalization for from two to four weeks for strict ketogenic diet. If the same results can be accomplished by a simple acid medication which can be taken at home, there may possibly be a future for this method of therapy; but here again it is not the method of choice in the aerogenes type. Fortunately, however, the aerogenes type, which so stubbornly resists treatment, only constitutes about 25 per cent of the cases, 75 per cent falling into the Escherichia coli classification.

Dr. Louis Bauman: I am not a urologist, but a medical man and a bit of a chemist, perhaps. I have tried to cover the literature on the ketogenic diet, and find that in the main the work is all confirmatory. Especially in England have there been very enthusiastic reports. One gentleman by the name of Wilson, I believe a urologist, suffered from pyelocystitis himself and was unsuccessfully treated by other methods. His urine cleared up in six days on the ketogenic diet and has remained clear without recurrence for eight months. He thinks that the treatment can only be carried out in a hospital; that carbohydrates should not exceed 20 grams; and that the ketosis must be maintained for at least a week after the urine is sterile. He had several cases in which there was a recurrence, and a repetition of the diet sufficed to re-sterilize the urine.

There seems to be no question with regard to the bacteriostatic properties of beta hy-

droxybutric acid at a pH of 5 or slightly over. As the ionization of beta hydroxybutyric acid at a pH of 5 is slight, while that of its sodium salt, which is formed in a more alkaline reaction is greater, the bacteriostatic effect is not due to the hydroxybutyrate ion. A point that has been emphasized by Dr. Clark and others is that the urine must be of a certain acidity, with a pH around 5 or slightly over.

In another larger group of pyelocystitis cases reported from England, fifteen of the most intractible were selected and put on the diet. For the first time a certain number of these passed sterile urine.

Many neurologists have had experience with a high fat diet in the treatment of epilepsy. About twenty years ago I worked with this diet in children, with the idea of studying acidosis. In children acidosis is produced with great ease because apparently they have very little glycogen supply in the liver. Acidosis is much more difficult to produce in adults. The first requisite, of course, is a good stomach. A dyspeptic cannot tolerate a one-sided diet containing more than 200 grams of fat. The patient must be responsive to the fat. In certain individuals it is difficult to bring the urine down to the proper acidity.

As to the use of ammonium chloride: It is probably the best acidifier of urine. We often give 6 grams a day which is tolerated pretty well. It is about one-ninth of a molecule. The ammonia is said to be converted into the neutral substance urea, so that we really furnish hydrochloric acid to the system. Six grams of ammonium chloride furnishes over 1000 c.c. of 0.1 normal hydrochloric acid. There is nothing you can conveniently give which will touch that.

I do not believe that the drawbacks to the ketogenic diet are serious. They are individual drawbacks, but not serious enough to influence the use of the method in general. In the Urological Department of the Presbyterian Hospital we have been studying acidification of the urine, and we plan to study ketone formation both in vitro and in vivo. The results thus far are encouraging, but not sufficiently conclusive to warrant an opinion. It is obvious that this is a new field which will require considerable clinical and laboratory study before its possibilities can be defined. But I believe that Dr. Clark and Dr. Helmholz are to be congratulated on this achievement which promises to be a real step forward in urology.

John A. Killian: What I gather from Dr. Clark's paper tonight and from his publications in the literature concerning the ketogenic diet in the treatment of urinary infections is that it effects a two-fold purpose—an increase in the acidity of the urine and an increase in the ketone bodies among which, according to Fuller, the bacteriostatic agent is beta hydroxybutyric acid. According to Dr. Clark it is necessary at times to supplement the ketogenic diet with acid salts. This suggests an interesting speculation. Ousterhout has recently demonstrated that acidity facilitates penetration through living membranes. It may be that the acidity of the urine not only maintains beta hydroxybutyric acid in free form (which is apparently essential for bacteriostasis) but also facilitates penetration of the bacteriostatic agent to regions beneath the surface of the lining membranes.

Dr. Clark has emphasized the necessity of determining the pH of the urine. In the normal individual on an average diet, the reaction of the urine varies throughout the day, particularly in response to the food intake. Although I have seen no data on the subject, it would be interesting to determine whether or not the ketogenic diet was accompanied by similar fluctuations in the pH. We know that the changes in the reaction of the urine are in some way associated with the secretion of hydrochloric acid by the stomach mucosa; we also know that fat stimulates the secretion of very little hydrochloric acid. It may be that with the ketogenic diet these fluctuations in the pH throughout the day are not as great as with an average diet.

Dr. Clark's statement that some cases will not develop a ketonuria although on rigid ketogenic diet, is interesting in relation to the reports of Murlin and his colleagues, on experiments on seven subjects on a high fat diet. In this small group there were some who showed respiratory quotients below the theoretical for fat oxidation. These quotients bore no relation to the ketonuria. This suggested that the fat was being converted into carbohydrates, and probably rendered available for oxidation later.

In reading the literature on the ketogenic diet applied to the treatment of urinary infections I was surprised to learn that apparently no control was exercised over the alkali reserve of the blood plasma. Apparently control is

limited to an analysis of the urine. Dr. Clark mentions experiments carried out on a group of experimental subjects in whom an average fall of 14 per cent in CO_2 combining power in five days was noted. Apparently he does not regard this fall in the CO_2 combining power as dangerous. For an experimental subject, a decrease in the alkaline reserve of the blood plasma of 14 per cent is probably not dangerous. However, a decrease of this amount in the CO_2 combining power of the blood plasma may be dangerous if the patient has impaired renal function, particularly if the initial CO_2 combining power is at the lower normal level or below the normal of 50 volumes per cent. It seems to me that the necessity of controlling alkaline reserve of blood plasma should be emphasized, particularly when the ketogenic diet is supplemented by the use of salts which in themselves have the power to decrease the alkaline reserve of the blood.

Dr. Nathaniel P. Rathbun: I think the ketogenic diet is an important adjuvant in the treatment of urinary infections provided one selects the proper cases. But I agree with Dr. Crance that we must first study our patients for gross urinary lesions. It does not seem to make sense that we can control cases with large stones until we have first removed them. I agree with Dr. Clark in his emphasis on the fact that it is important to determine the presence of and eliminate focal infections, as we do in prostatitis.

Having eliminated these infections of the urinary tract and the focal infections—the cases of colon bacillus infection of the urinary tract—I believe the ketogenic diet works wonders. I have also had some failures. I think that, as Dr. Clark stresses in former papers, it is apt to fail unless we remember that it is very important to determine the group of colon bacillus with which we are dealing. I agree with his former conclusions that better results are obtained in the escherichia group, and little effect in the aerogenes. But that is too difficult a question to thrash out now, for most laboratories are working short-handed, with few technicians.

I have had a number of failures with the ketogenic diet because while we got the pH down to the required amount, we could not develop a ketosis. There are patients who cannot tolerate the ketogenic diet. It keeps them nauseated and sick. In those cases in which a ketosis can be produced and the pH kept down, dramatic results have been obtained in a fair number of cases.

I should like to ask Dr. Clark whether he has ever used the diet in very young infants. My friends among the pediatricians have been rather fearful of using it, say in infants ten to eleven months old with pyelitis.

Another point: In the female children, or even female adults, Dr. Clark mentioned a few cases in which the infection was cured in the kidney but persisted in the bladder. There are two possibilities here: The first is that of residual urine; the second is that of infections in the urethra. These may be factors in continuing infection in the bladder.

Certainly ammonium chloride sounds like the ideal drug for producing acid urine. I like sodium benzoate, though. One or two drams daily often produces a highly acid urine when other drugs fail.

I think we should remember that there are other weapons in our hands for the treatment of chronic obstructive infections. I certainly would mention among these the use of bacteriophage. Here we have sometimes the same result as with the ketogenic diet. I have a feeling that a properly made phage will without question kill any organism it comes in contact with. The point is that most of our cases are of the so-called pyelitis or pyelonephritis variety, and therefore there is no contact with the organism. One of the important points of the ketogenic diet is that it gets into the blood stream, and no matter where the infection is it gets to it.

I highly subscribe to the lack of faith expressed in urinary antiseptics. I have had little results with them. If I use any at all, I feel that the old formine, or urotropine, is as good as any.

In conclusion, it is only fair to say that I have seen any number of cases of chronic colon bacillus infection of the urinary tract producing practically no symptoms and in which no form of treatment—urinary antiseptics, ketogenic diets or bacteriophage—has helped. After giving these things a reasonable trial, I am disposed to let these patients carry on in their more or less happy state of misery.

Dr. Stanley L. Wang: Dr. Clark mentioned quartz light therapy in cases of tuberculosis of the bladder. We have used it rather extensively at the Department of Urology (James Buchanan Brady Foundation), New York

Hospital. In about 40 per cent of the cases treated it has been the most useful procedure, and effects beneficial results. We have used it with a specially constructed applicator; irradiating the interior of the bladder over a long period of time.

The ketogenic diet has in our experience been useful in the treatment of colon infections of the urinary tract. We have used it for some time, following the methods introduced by Dr. Clark, and the results have been encouraging. It needs to be carried out precisely to obtain satisfactory results; that is, the diet must produce the ketone substance in the urine, and the acidity must be brought to the proper point. Forty per cent cream and eggs are most effectual of the various foods. The effect is so profound at times that it suggests that the usual ketogenic diet of 3.5 to 1 might be started at a lower ratio and increased. In some patients a low sugar diet for a day before starting the ketogenic diet is worth while. The colon bacilli often disappear in a striking manner. Our difficulty has been that the infection may recur when the patient discontinues the diet. The plan of recurrently administering the diet has proved more satisfactory, however, than continuing it for a long time.

The main objection to the diet is the discomfort caused from the ingestion of the necessary large amounts of fat. For some time we have been seeking a method of remedying this objection, very real to the patient. We have been considering the method of intravenous fat injection, but it seems doubtful at present that this would offer an effectual means of introducing sufficient fat for the purpose, particularly in adults. Increasing a less rigorous diet with fat in concentrated form seems rather the angle to work from.

The ketogenic method of treating urinary infections is very interesting, for it proves that the urinary acidity can be accurately changed in most instances to the point where the bactericidal substances in the urine are made most active. The implications are quite obvious.

We have not used the diet in any of our tuberculosis cases, as it seemed too strenuous for them.

Dr. Anson L. Clark (*Closing*): I believe Dr. Helmholz has had one of the companies specializing in baby foods prepare a powdered food with a high fat content which he has been able to use successfully in babies with bacillary infections of the urinary tract. Personally I have had no experience in its use.

We all realize the importance of a complete urologic examination where the patient is having the characteristic symptoms of the urinary tract infections. I should like to make an emphatic plea for control of the infecting organism before the patient is subjected to much instrumentation. In many instances the acute symptoms following cystoscopy and ureteral catheterization would be avoided if the colon bacillus was first controlled by having the ketone bodies in the urine before introducing the instruments.

Following operative procedures such as prostatic resection, when the operative site has entirely healed this type of therapy has been beneficial in clearing up the infection. In the presence of blood or sloughing tissue, which probably affords a better culture medium for the organism, the ketogenic treatment is probably not indicated. The ketone bodies are a very mild bacteriostat c agent, and more satisfactory results will be obtained if the patient is treated symptomatically until healing has been accomplished.

CRITICAL ANALYSIS OF 35 DEATHS FOLLOWING APPENDICITIS

WHEN SHOULD DELAYED OPERATION OF RUPTURED APPENDICITIS AND PERITONITIS BE USED?

C. R. DAVIS, M.D., F.A.C.S.

Attending Surgeon, Miriam Memorial Branch of Grace Hospital; Chief of Surgical Staff, Parkside Hospital

DETROIT, MICH.

IN reviewing 35 deaths which have occurred in a consecutive series of 1130 cases of appendicitis in which I have operated, I have endeavored to find out which patients might have been saved, could I have a second opportunity to take care of them. Because articles advocating delayed operation for appendicitis at certain stages of an attack have appeared with greater frequency recently than for many years, naturally my interest has been aroused again in this form of treatment. In reviewing mortality rates and case reports to find out if delayed operation in certain cases really gives a lower mortality than immediate operation in all cases, I found that statistics on death rates did not convince me that delayed operation has any advantage over immediate operation, but many of the case reports are convincing that delayed operation has value under certain conditions. When I attempted to tabulate the conditions when operation should be delayed, I found disagreement among surgeons which causes confusion that should be cleared up before delayed operation can be practiced intelligently and used universally. Some form of peritonitis is the chief indication for delaying operation, but the main differences of opinion are whether it should be spreading or generalized peritonitis. While reviewing the writings of Ochsner, Sherren and others, I became convinced that some who advocate delaying operation are not familiar with the methods which have been used, and that others who advocate delaying operation cannot, or at least do not, give the indications and contraindications for doing so. For these reasons I shall discuss delayed operation of appendicitis, repeating the original methods, and give the indications and contraindications for its use, stating ideas of other writers and my own. I shall then cite the various mortality rates which interested me and finally give an analysis of my own deaths.

The delayed operative treatment of appendicitis, generally known as the Ochsner treatment, frequently is called the Ochsner-Sherren treatment; Ochsner of this country originated the treatment over thirty years ago; Sherren of England used a similar treatment. Because the treatments used by these two men are not identical, delayed operative treatment should not be called the Ochsner-Sherren treatment, but the particular form of treatment used by surgeons should be called by the name of the particular man whose treatment has been followed in each case.

The Ochsner[6,11] requirements are: The stomach must be emptied of contents already there by lavage at once; lavage may be repeated from one to three times. He later modified the use of lavage so that lavage should be done whenever nausea and vomiting occurred. He recommended a particular method for doing lavage; spray the throat with 4 per cent cocaine repeatedly for five minutes, turn the patient on the right side, support the head and shoulders with pillows, and wash out the stomach with saline at 100°.[9] At first he

had the patient rest in bed with the shoulders slightly elevated, but later he had the head of the bed raised 18 inches. His first reports required enemas of 1 ounce of concentrated predigested foods with 3 ounces of normal saline to be given every four hours. Later he preferred 500 c.c. of saline with 50 c.c. of glucose and 2 c.c. of deodorized tincture of opium to be given by proctoclysis and to be repeated after two hours. For rest he gave atropine, grain $\frac{1}{150}$, and morphine, grain $\frac{1}{4}$, to be repeated when necessary. He continued the treatment until the patient had the general appearance of getting better, when gas began to pass, and when the signs of general peritonitis had gone. He later decided to continue the treatment until the patient had been normal for four days, after which he began feeding the patient. He operated only when the operation could be done safely; he opened abscesses under local anesthesia; he operated upon all acute cases during the first thirty-six hours, and in cases of superficial circumscribed abscess where the disease had persisted longer than thirty-six hours. Ochsner gave absolutely nothing by mouth which is importantly different from Sherren's procedure.

Sherren[10] gave water by mouth, as much as was needed except when vomiting occurred repeatedly, when he ordered lavage and continuous proctoclysis. He used Fowler's position and gave morphine for pain. After the pulse and temperature had settled, he gave more nourishment by mouth and ordered an enema. If there was no improvement, he drained only; unless the appendix presented itself, whereupon he removed it as well. If an abscess formed, he opened it where it pointed, rectally if necessary, and did an appendectomy three months later.

Either the Ochsner or the Sherren treatment could be modernized without changing the underlying principles. A continuous Levine nasal tube might be substituted for the repeated gastric lavages, and parenteral fluids consisting of saline and glucose in large amounts for proctoclysis. The rest and elevation of the head of the bed should be the same. If I were to delay operation in any of my cases, it would be with these modifications of procedure. I prefer the Ochsner edict of giving nothing by mouth to the Sherren edict of fluids as much as needed.

In discussing the indications and contraindications for delaying operation I shall begin by again quoting the original advocates of the treatment. Ochsner said that there are two classes of patients who are not satisfactory for delayed operation; the very young and the very old; the very young because there is no omentum to wall off pus and because the child cannot be kept quiet and will not submit to the treatment readily; the very old because they do not stand confinement in bed and a restricted diet. I have found no author opposed to these views. Ochsner operated upon all acute cases during the first thirty-six hours and in cases of superficial circumscribed abscess where the disease had persisted longer than thirty-six hours. In this respect Ochsner's viewpoint about coincides with that of Sherren.

Hamilton Bailey[1] states that delayed operative treatment "may be strongly recommended as a routine practice in cases of appendicitis of over forty-eight to fifty hours' duration . . ." and:

> With the following exceptions when immediate operation is advisable.
> 1. In the presence of hyperesthesia (which indicates that the appendix is still unruptured).
> 2. Where there is obvious general peritonitis.
> 3. In children under eight.
> 4. Where a strong purgative has recently been ingested, and
> 5. Where there is uncertainty in diagnosis, particularly when perforated diverticulitis is the alternative.
>
> In a civilized country it should be criminal to attempt this treatment outside a surgical hospital.

I shall not quote all of the other necessary details upon which Bailey insists but shall refer readers to Bailey's book for them.

Potter and Coller[7] state that "since 1931, the delayed operation has become in our clinic practically routine in instances where it is felt the process has extended from the right iliac fossa to the general peritoneal cavity. . . . " They consider "the forty-eight hour period the most probable length of time before perforation occurs" although they have delayed operation in cases of obvious peritonitis less than twenty-four hours after onset and have promptly operated upon patients sixty hours or more after the initial symptoms. Diffusing peritonitis appears to be their chief indication for delaying operation. I, also, believe that the condition present and not the time element should rule.

In comparing the opinions and case reports of the various authors, I find that Ochsner and Sherren evidently practiced delayed operation in general peritonitis; Bailey does not; Potter and Coller evidently do, but they do not make the point entirely clear, leaving me wondering if they delay operation when peritonitis is just beginning or wait until it is well advanced, or both. I favor Hamilton Bailey's opinion and believe that delayed operation can accomplish nothing in general peritonitis because I cannot believe that in general peritonitis the pus will run down to the right lower quadrant and localize, but will pocket in many places, as I have seen in necropsies on patients who have died of general peritonitis. According to case reports which I have seen of patients who have died after delaying operation, multiple abscesses occurred in many of them also. In spite of my agreement with Dr. Bailey, I am inclined to want to let the results in these bad cases of peritonitis be absorbed by delayed operative treatment rather than by immediate operative treatment, chiefly because immediate operative treatment has so little to offer. Because all advocates agree that it is only appendicitis with some form of peritonitis in which delayed operation should be used, I shall consider in my discussions only cases of appendicitis with some degree of peritonitis as suitable for delayed operative treatment.

Another point unsettled is what constitutes age limits. Old and young are exempted from delayed operative treatment, but Hamilton Bailey is the only one who gives a specific age, eight for the young, but even he does not mention the old age limit. According to the statistics of Keyes[5] ten should be the maximum age limit for the young and sixty the minimum age limit for the old. Considering the anatomy of youth and the degenerations of the old, I shall establish these specific limits as my guide in discussing my cases. Dr. Keyes gives these figures:

TABLE 1

Ages	*Cases of Appendicitis*	*Deaths*	*Mortality (per cent)*
All	1859	62	3.33
1 to 9	122	15	12.29
50 to 59	49	4	8.16
60 to 76	19	8	42.11

The percentages are definitely higher and out of proportion for those under ten and over sixty. Even over fifty the percentages are considerably higher, but they are so much higher over sixty that I shall use sixty as my minimum age limit in the old.

As to whether the mortality rates for delayed operation are really lower than rates for immediate operation, my study of case reports convinces me that lives have been saved by delayed operation which I believe might not have been saved by immediate operation, but, of course, ultimate percentages tell the true story. Here are a few picked statistics for delayed operation: "5.10 per cent[3] at Manchester Royal Hospital, 4.12 per cent at St. Thomas Hospital, and 3.5 per cent at the London Hospital" quotations which are accompanied by the remark that those "mortality rates, however, occur in selective cases free from any loading by those dangerous, fulminating, well-nigh hopeless cases in which fatal termination is imminent . . . and which surgery absorbs." These rates, of course, are not below the average. Guerry[4] cites 128 cases in which he deferred operation with a mortality of 1½ per cent,

although he said that the Ochsner method has frequently been supplemented by simple incision and drainage. The cases in which incision and drainage were used should be classified under various forms of operative technique instead of under delayed operation, and the mortality rate should be listed under immediate operation. The rates just cited have been quoted frequently by advocates of delayed operation, but the rates are as high if not higher than those of many surgeons who operate immediately upon all cases of appendicitis. If picked statistics are to be compared, many more records of low mortality with immediate operation can be found which compare very well with the fewer records which can be cited by the most ardent advocates of delayed operation. For example, Cottle[2] cites 12,204 cases in all naval stations during a ten year period, 7875 of which were acute, with a mortality of 1 per cent. Quain and Waldschmidt[8] cited 1000 cases with a mortality of 2.7 per cent after having had unfortunate experiences with delayed operation. Greenwood[3] had 206 cases, 18 per cent of which had general peritonitis with no deaths. It is easy to see that writers can readily prove their points by statistics, but I must confess that it appears to me that on the whole immediate operation has the edge over delayed operation. Assuming that lives can be saved by delayed operation and keeping in mind the indefiniteness about when operation should be delayed, I shall now review my own deaths to see if any and how many of those deaths could have been prevented by different treatment and shall state by which treatment I think the deaths could have been prevented; but I shall not cite serious cases which have recovered after immediate operation to see how many might have died had I tried delaying the operation. I shall try to fit my cases into groups governed by the foregoing rules and exceptions, first classifying them according to ages:

Of the 35 patients who died the ages were as follows:

Table II

Age	*Deaths*
1 to 10 inclusive	8
11 to 16	2
17 to 30	6
30 to 49	10
50 to 59	7
60 to 69	1
70 to 90	1

For the sake of brevity I shall tabulate the remaining information about these cases according to the indications and contraindications for delaying operation, deleting the detailed report:

Table III

THIRTY-FIVE DEATHS FROM APPENDICITIS IN 1130 OPERATIONS, CLASSIFIED ACCORDING TO INDICATIONS AND CONTRAINDICATIONS FOR DELAYED TREATMENT

1. *Contraindications:*	
A. Age*	10
B. Local abscess†	2
C. Chronic appendicitis	1
D. Inability to diagnose correctly	5
E. Time limit less than 48 hours‡	4
F. Fulminating	2
G. Strong cathartics	1
2. *Indications:*	
A. Possible better results from delayed treatment	4
B. Outcome with delayed operative treatment possibly the same§	5
C. Outcome better with either delayed treatment or a more clever operation	1

* In children and the old I now use more fluids and glucose than formerly. In the two old patients, one death five months after the onset of peritonitis resulted from voluntary starvation and could have been prevented by forced feeding by tube or by the use of insulin; the other death was aided by inaccuracy in diagnosis, the diagnosis being intestinal obstruction from carcinoma of the bowel. The sickness had persisted for two weeks.

† One died from the anesthetic (spinal). One died from lack of cleverness of operation.

‡ One of these early patients died of apoplexy on the third day.

§ One patient recovered from peritonitis and died more than six weeks later after an operation for retroperitoneal abscesses in the mesentery, a complication. A second patient died from cerebral embolism from thrombosis in the femoral vein. A third patient had been sick twelve days, improved daily for five days after operation and died from exhaustion with a clean peritoneum. A fourth patient had a leucocyte count of 4500 with 91 per cent polymorphonuclears. The fifth patient was a very alcoholic and fat man.

Of the 10 cases which are classified under Indications, in only 4 cases will I admit that results might have been improved by delaying operation. Whether it is 4 or

10 cases upon which we could have delayed operation, at best the variation in mortality would not have been more than 1 per cent. Had I delayed operation in the maximum number of suitable cases, I would have done so in 12 cases, the other 2 being the fulminating cases. Had I used delayed operation in the minimum number of cases, I would have done so in 4 cases which averages from 1 in 94 cases to 1 in 282 cases, or one case a year for the busy surgeon and one in three years for the average surgeon. Although delaying operation may occasionally save a patient's life, it is hard to know what case to select for the delayed operative treatment with such long intervals between cases. With the statistics so evenly balanced I fear teaching delayed operative treatment, because its misapplications may result in an ultimate higher mortality. As an example I shall mention a colleague's experience. After having delayed operation for several days in one of his patients, he operated and found an unruptured appendix and no peritonitis.

Listing the cases which I think I might have saved with a second opportunity by more clever handling or with care based upon more recently acquired knowledge, I find there is a possibility that I might have saved 8 or possibly 10, which also would have reduced the mortality slightly less than 1 per cent. Thus, it appears that whether I delayed operation, or corrected the errors in treatment, the reduction in mortality might have been about 1 per cent, and by doing both I believe that the reduction in mortality would have been no greater, because the reduction would have been made by either method in the same cases. With the ultimate results so evenly balanced I doubt the wisdom of continuing confusion in professional minds about delayed operation until such time that definite and specific instructions can be given in such a way that we all can follow the same procedures intelligently and know just when to practice delaying operation. For the present we can gain more by making more accurate diagnoses, operating more cleverly in difficult cases, and earlier in all cases. If we must delay operating on any cases, I favor delay only in fulminating cases. Whatever we do, we should never forget what Osler said: "The general practitioner does well to remember—whether his leanings are toward the conservative or radical methods of treatment—that the surgeon is often called too late, never too early."

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ACUTE APPENDICITIS COMPLICATED BY PERITONITIS*

IMMEDIATE AND LATE RESULTS IN 126 CONSECUTIVE CASES

GEORGE W. KEHL, M.D., F.A.C.S. AND CALVIN B. RENTSCHLER, M.D., F.A.C.S.

Surgeon, Reading Hospital — Associate Surgeon, Reading Hospital

READING, PA.

THIS article is based on an analysis of the immediate and late results of 126 cases of acute appendicitis, complicated by peritonitis, surgically treated at the Reading Hospital, from January 1, 1929 to January 1, 1935.

The data, covering the end-results, were obtained by a questionnaire sent the patient, and the information was received from the patient, a member of the immediate family, or from the family physician. The questionnaire was sent January 1, 1935.

Any disease, which causes death of as many people as appendicitis, warrants careful scrutiny of the management of these cases with probable improvement in the methods of treatment.

If all types of appendicitis are considered as one group, we will have a much too optimistic picture of the disease. It is known to all surgeons that the so-called clean cases have a negligible mortality. If this group is segregated, the remaining "drainage" group constitutes the serious cases in which the mortality has been ranging from 10–25 per cent, according to the present writers on the subject. It is on this group that our attention should be concentrated if we expect to reduce the morbidity and lower the mortality in appendicitis.

DIAGNOSIS

The diagnosis depends chiefly on a good history and careful physical examination. Here, of all places, a thing not done well will exact its toll in terms of morbidity and mortality. The historian must go back to the very beginning of trouble, starting from the time the patient first suspected something wrong. Every symptom and complaint, and their changing character, must be carefully evaluated. The type and amount of intake must be ascertained. The time of seeking medical aid should be inquired. There can be no doubt that many patients do not notice the earlier symptoms, or attribute them to dietary indiscretions, and first seek medical aid when the appendix has already perforated with resultant abscess formation or peritonitis. Many of the patients in the present series did not call a physician until too late.

When the symptoms and physical findings are typical, the diagnosis of appendicitis is relatively simple and certain. Pain and tenderness in the lower right quadrant usually is appendicitis. Pain, tenderness, rigidity in the lower right quadrant, with elevated temperature, pulse and leucocyte count, with nausea and vomiting, usually is acute gangrenous or suppurative appendicitis, and this is often complicated by localized peritonitis. Often, however, the symptoms are not typical, or the disease has advanced very rapidly, or the physician has been called too late, and under such circumstances, the diagnosis may be extremely difficult. The longer the patient goes without a diagnosis having been made, the more difficult the diagnosis. The disease advances with varying degrees of rapidity; some cases are no further advanced in three days than others in twenty-four hours. Some have spreading peritonitis

* From one of the general surgical services, Reading Hospital. Read before the Berks County Medical Society.

in a day; others, even after a week, have little more than a lymphatic exudate around the appendix. When the disease has advanced to the stage where the resultant complications are well developed, the clinical picture not infrequently becomes mysteriously bizarre. Thus, we may find marked distention of the abdomen with continuous reversed peristalsis, as in advanced primary intestinal obstruction; or the patient may present the picture of shock, cold extremities, rapid pulse and respiration, etc., the result of a perforated appendix with generalized peritonitis.

In cases of appendicitis with abscess formation, the abscess mass can usually be palpated by careful abdominal, pelvic and rectal examination.

In the case of spreading or diffuse peritonitis, the diagnosis is often extremely difficult and may resolve itself into the differential diagnosis of an acute surgical abdomen. Here one must think of adhesions with obstructive symptoms, ovarian cyst twisted on its pedicle, ectopic pregnancy, Meckel's diverticulum, ileocolic intussusception, acute salpingitis, slowly leaking duodenal ulcer, low acute disease of the gall bladder, etc. A careful history will aid much in the differentiation of these conditions; however, if none of these conditions could be definitely ruled out, it must be remembered that these lesions are all surgical in treatment.

TREATMENT

Long ago, Osler said: "There is no medical treatment for appendicitis." That dictum is as true today as it was then. The practice of giving a good cathartic, or of "freezing the attack," or finally, the policy of waiting to be certain, before hospitalization, or at least surgical consultation, has without doubt cost many a life which could have been saved. It is much better to move a case into the hospital before it is decided on operation is absolutely necessary, than to wait until too late. Mistakes will be made, but it is much better to sacrifice an occasional innocent, though suspected appendix, than a human life that might have been saved by timely operation. Furthermore, by removing even a normal appendix we have, at least, insured the patient against the disease in later life.

Not infrequently the patient or the family refuses hospitalization. This type of mental attitude can usually be improved in one of two ways; first, by insisting on a consultation, and in this particular type of illness, preferably with a surgeon; and, second, by absolutely resigning from the case. I believe, if diplomatically and sympathetically done, at least, the latter of these alternatives need rarely be used. This is a matter of life or death. Every physician knows that in this disease delayed surgical treatment is by far the chief cause of death.

Most of these patients were brought into the hospital in an extremely ill condition. Often, they were brought from the extreme ends of the county. Immediate steps were taken to get the patient ready for operation. In no case was the deferred method of treatment employed. We feel that too many factors, of which one can have no preoperative knowledge, are concerned. Thus, in the matter of diagnosis, one can never be 100 per cent certain of the exact differential diagnosis. In some cases, with a mistaken diagnosis, delay would do no harm; however, in others, such an intussusception of the ileocolic type, or in a slowly leaking duodenal ulcer, disastrous results would have to follow if such patients were treated along expectant lines. Furthermore, as Eliason[1] states, some cases may present the clinical picture of generalized peritonitis: generalized abdominal rigidity, abdominal silence, with high temperature and high leucocyte count, and yet, at operation, it will be found that the appendix is not even perforated. Then too, many of these patients have been violently purged before their arrival at the hospital, and we agree with Jeff Miller[2] that time is always lost in waiting to see what the purge is going to achieve.

Table 1
AGE INCIDENCE IN 126 CASES REPORTED

Age	*Total No. of Cases*	*Deaths*
Birth to 5 years	6	1
6 to 10 years	27	1
11 to 20 years	41	2
21 to 30 years	16	4
31 to 40 years	10	0
41 to 50 years	7	2
51 to 60 years	10	3
61 to 70 years	3	1
71 to 80 years	6	2

OPERATION

At operation, the cases were classified into three groups:

1. Acute gangrenous or suppurative appendicitis, with localized peritonitis. A perforation is usually seen. There is free pus about the appendix area. Often the appendix lies in a purulent bleeding bed with the adjacent structures acutely inflamed. Occasionally, perforation cannot be detected, but with the appendix gangrenous or suppurative, and lying in a bed of purulent fluid, one must remember that bacterial penetration of a necrotic appendix can produce as profound an effect as actual rupture of the wall. This group comprised 18 cases, with 2 deaths.

2. Acute suppurative appendicitis, with abscess formation. There were 41 cases in this group, with 2 deaths. In 8 of these cases, the abscess was merely drained without removing the appendix. One of them later required an appendectomy, but 7 had no further trouble.

3. Acute suppurative appendicitis, with spreading or generalized peritonitis. There were 67 cases in this group, with 12 deaths. All of these patients were extremely ill. Many of these had much free pus with extensive inflammation of the peritoneum and bowel serosa; others seemed older, with the coils of bowel extensively adherent, with many small pockets of pus, and the intestines red and dull.

A right rectus incision was always employed, with the exception of several cases where the operation was performed for appendiceal abscess. As gently as possible, the omentum and small bowel were pushed to the left, and the cecum located. The appendix was located next, and usually was removed. The clamp and cautery method, with inversion of the stump, was employed. At times the cecum was so firm and indurated that inversion of the stump was not practical. Surgical judgment is quite unanimous that the appendix should practically always be removed, except occasionally, in cases of appendiceal abscess. McGrath and Eiss,[3] and others, have shown that the late postoperative complications in acute suppurative appendicitis are more severe and more frequent in those cases where appendectomy was not part of the operation.

In the cases where abscess formation was present, the abscess was carefully drained without more dissemination of the infectious material than could be helped. Very little pressure, if any, was used in walling off the infected area. In those instances where the abscess was not opened by the incision, a point of cleavage was easily found. Abscesses varied from a small mass of pus, surrounded by inflammatory structures, usually omentum, parietal peritoneum and coils of small bowel, to extensive abscesses filling a major portion of the lower half of the abdomen. In some such cases the appendix was not even looked for.

In the cases of diffuse or spreading peritonitis, the appendix was removed in practically every instance. The free pus, variable in amount, was aspirated. In no instance was the abdominal cavity irrigated. The extent of the spreading or generalized peritonitis can seldom be accurately ascertained because to do so would mean unnecessary enlarging of the incision and abdominal exploration.

In all cases of peritonitis, two Penrose drains, as well as a soft rubber tube drain were inserted into the pelvis, and often, a Penrose drain was inserted upward toward the liver, and in several instances one, or

more, toward the midline, if indicated. Every effort is made to keep all the drains lateral to the coils of the intestines. Aside from the series of cases under discussion, all of which were "drainage" cases, there were encountered during the same period many cases of the gangrenous or suppurative type, with yellow plastic exudate on the outside of the appendix, and with varying amounts of serous fluid in the abdominal cavity, but, in these cases, the abdomen was sewn up tight. None of these patients died; however, 2 of them developed abscesses, which were subsequently drained, with prompt and complete recovery. No case of appendicitis, with peritonitis, was deliberately closed without drainage.

There are sporadic attempts over the country to treat appendicitis complicated by peritonitis by performing an appendectomy and closing the abdomen tight.

Colt and Morrison[4] report that a statistical analysis of 1408 cases of acute appendicitis fails to justify any conclusion for or against drainage of the peritoneal cavity.

Guerry,[5] whose statistics cover a surgical experience of many years, and which are also the best on record to my knowledge, states that Taite's original rule of "when in doubt, drain," changed to "when in doubt, don't drain," may be witty, after a fashion, but that it is altogether vicious as a surgical principle.

Gastrointestinal tract drainage, by means of enterostomy (usually ileostomy), cecostomy or appendicostomy, is employed throughout the country. Some advocate that it should be done at the time of the primary operation.

Easton and Watson[6] employ it to forestall the onset of postoperative paralytic ileus and to prevent obstruction from adhesions by keeping the intestine in normal motion and allowing prompt escape of gas and fluid. They report good results. These procedures are employed by others in the treatment of postoperative obstruction. A cecostomy was performed on one of the patients in the present series, with complete recovery. One other patient should have had an enterostomy, but it was delayed too long. Postmortem examination revealed complete intestinal obstruction secondary to plastic peritonitis.

POSTOPERATIVE TREATMENT

The surgeon himself must be willing, at least, to thoroughly supervise the postoperative course. These patients are desperately ill. Complications should be discovered in their incipiency, when they can be most successfully combated. A stomach tube, an intravenous infusion, or a hypodermoclysis, when needed, is worth a hundred times more when used early than the same measure used late, or as a last resort.

Postoperatively, the Ochsner treatment, in a general way, was employed in all cases. The patients were put in the Fowler or modified Fowler position, enough morphine sulphate was given, and fluids were administered, preferably by hypodermoclysis. When absorption by hypodermoclysis seemed inadequate, fluids were given intravenously in the form of glucose and saline solutions. Fluids by mouth were not started before twenty-four to forty-eight hours, depending on the severity of the illness. Blood transfusions were freely employed in debilitated and the more toxic patients, using both the direct and indirect methods, more often the former.

In all cases where vomiting occurred, the stomach was lavaged by the interrupted method. More recently, the gastroduodenal aspiration method, as revived by Wagensteen,[7] was employed in some of the cases. Soapsuds enemas were given, but usually not before the end of the third full postoperative day. Pituitrin and eserine were never given. Catharsis was not resorted to until all abdominal drains had been removed, and then only in mild form. Drains were not disturbed for six days; they were then gently loosened and removed the following day. Light re-packing of the tract was always done. The

wound was kept open in this way for a varying period of from two to three weeks, depending upon the particular case.

Table II
COMPARATIVE MORTALITY OF SERIES OF VARIOUS AUTHORS*

Series	*Mortality (per cent)*
Arnheim and Neuhof[8]	14.5
Muller[9]	9.0
Walker[10]	15.1
Ashhurst[11]	13.7
Bower[12]	12.2
Keyes[13]	12.1
Davis[14]	13.9
Kehl and Rentschler	12.6
Dixon[15]	9.3

*The statistics were not all based on the same premises.

IMMEDIATE RESULTS

In this series of 126 cases, 110 patients survived and 16 died, a mortality of 12.6 per cent.

Among the group that survived, several developed rather serious postoperative complications. There was one instance of an alarming, although non-fatal, pulmonary embolism, secondary to a femoral, probably also iliac, thrombophlebitis. One patient developed residual abscesses, requiring first a right, then a left colpotomy, and finally, a secondary abdominal incision and drainage. Another patient developed an acute pyelitis. A pleural effusion, apparently non-specific, prolonged the convalescence in one case. Three patients developed temporary fecal fistulas; two, of two weeks' duration, and one, of two months' duration. The postoperative course of the remainder in this group was practically uneventful. All of them were discharged from the hospital in excellent condition, all drains removed, and the wounds almost, if not entirely, closed. The average stay in the hospital for the 110 who survived, was 28.5 days.

Of the 16 who died, 13 had postmortem examinations. All but 2 of these 16 had been given one or more cathartics, usually castor oil, before admission to the hospital. The average duration of illness before admission was four days. Every one of these 16 patients had acute generalized plastic or purulent peritonitis. Nine of the 16 had an additional complication, which was either directly responsible for the death or acted as the terminating factor in cases already desperate. In 3 instances, postoperative obstruction, 2 partial and 1 complete, secondary to the peritonitis, complicated the picture. Two patients were seventy-five years of age, and both had advanced arteriosclerosis with myocardial degeneration. In another case, acute cardiac dilatation was the terminating factor. One patient developed a large sub-diaphragmatic abscess postoperatively, and died after a secondary operation. In one case, extreme obesity was probably a factor, and one developed marked pulmonary edema and congestion.

LATE RESULTS

All of the 110 patients who survived have been traced. One hundred and seven state that they are generally well; however, 3 have incisional hernias, and another, according to the family physician, has a potential incisional hernia. Two patients were operated on for the repair of incisional hernias, and are now well. One was operated upon for adhesions and has had no further trouble.

Five described their condition as being fair, but did not mention any complaints. Several patients mentioned minor operations, such as tonsillectomies, and a few had minor complaints, none of which was relevant to their appendicitis or its management.

The remaining 3 patients, it was learned, had since died; one, as the result of pneumonia at seventy-four, another of cardiovascular disease at seventy-eight, and the third following an operation for exophthalmic goiter.

CONCLUSIONS

1. Surgical treatment of appendicitis before appendicitis is complicated by peritonitis is the preventive method of treatment, and by far the superior method.

2. The group of cases under consideration is a reflection of self-medication and procrastination before hospitalization, or at least, before surgical consultation.

3. The medical treatment of appendicitis, namely by ice-bags, drugs and particularly by catharsis, should be considered malpractice.

4. The treatment of appendicitis complicated by peritonitis is preoperative, operative and postoperative, all three of which are equally important. The treatment demands expert surgical judgment and surgical ability.

5. Except for a reasonable preoperative preparation, none of the patients in the series was subjected to deferred surgical treatment.

6. All the cases of appendicitis complicated by peritonitis received intra-abdominal drainage.

7. The immediate and late results of the patients who recovered are considered excellent.

8. In the present series of cases, the mortality is 12.6 per cent. Two of the patients who died were over seventy-five years of age.

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DUODENAL ULCER IN INFANCY AND CHILDHOOD

A CASE OF PERFORATED DUODENAL ULCER IN A CHILD OF SEVEN

KIKUWO TASHIRO, M.D., PH.D. AND NORMAN KOBAYASHI, M.D.

LOS ANGELES, CALIF.

THE occurrence of gastric and duodenal ulcers in infants and children, though conceded rare, is evidently much more common than is generally recognized. Less than forty years ago this condition, as affecting infants and children, was practically unknown. Since then there has been a steady but almost too unobtrusive accumulation of cases, and even today the report of such a case as this may still be regarded as a curiosity rather than as an imminent possibility.

As indicated by the literature, the incidence of gastric and duodenal ulcer is considerably higher among infants than among children. In 1903, Borland[1] described a case of duodenal ulcer in an infant of eight months suffering a severe eczema. Holt,[2] from a study of 95 cases of duodenal ulcer in infants under the age of one year, collected in 1913, stated that 70 per cent of those reported were observed between the ages of six weeks and five months, about 10 per cent occurring in the newborn. Until 1919 Theile[3] found recorded 119 gastric ulcers and 185 duodenal ulcers in patients under the age of sixteen. In 1922 Paterson[4] discovered about 100 cases of duodenal ulcer in infants in the literature, contributing 2 of his own.

Chronic ulcers of the stomach and duodenum are extremely rare before the age of puberty, this generally being attributed to the blandness of diet, lack of hyperchlorhydria, greater gastric motility, and the rapid regenerative ability of the gastric mucosa of children. Proctor,[5] after an extensive review of the literature up to 1924, found only 19 cases of chronic peptic ulcer in patients under the age of fourteen acceptable by his standards; he added 3 more. In this series there were 17 gastric ulcers, while the duodenal ulcers numbered only 5. Of the 17 gastric ulcers 5 perforated, 3 of these terminating fatally, while 2 patients were saved by surgery. Of the 5 duodenal ulcers only 1 perforated, reported by Bichat:[6] a girl of fourteen, who suddenly developed peritonitis after having had symptoms for three to four years; operation was followed by recovery.

Dickey[7] again reviewed the literature in 1926, finding 25 cases of duodenal ulcer in children, contributing 3. He revealed that of the 25 cases then recorded 5 were diagnosed at autopsy, 11 at operation, and only 9 were recognized clinically. Of the latter group several were confirmed at operation, the others being treated medically. Of the 25 cases, he points out, only 5 were diagnosed at any time as duodenal ulcer.

In 1932 Bloch, Bronstein, and Serby[8] presented 5 cases of chronic duodenal ulcer, which they observed in children between the ages of five and thirteen. The lesion was demonstrable by *x*-ray in all cases save one; in this case diagnosis was confirmed by surgery.

Literature upon this interesting subject has increased markedly, especially during the last decade, an indication, no doubt, that cases are being recognized with greater frequency. Evidently, and unfortunately, the accumulation had not yet gathered enough momentum to make the practitioner in general "pediatric peptic ulcer conscious." We have been unable to find on record more than 3 cases of perforated duodenal ulcer in children, with surgical intervention and recovery. In 1910 Bichat,[6] in France, recorded what we

believe to be the first case, in a girl of 14; in 1925 O'Flyn,[9] in England, reported his case, a boy of fourteen; and in 1927 Pototschnig,[10] in Italy, reported a case in a girl of eleven. Imfeld[11] also described a case in 1911; his patient, however, was a boy fifteen and one-half years of age.

We chanced upon this case of our report as a surgical emergency in a suburban practice, and the "postoperative realization" of its unique position has prompted the recording of this interesting experience. Incidentally enough, it appears to be the youngest such case on record, as well as the first to be recorded in America.

CASE REPORT

History of Illness: On the morning of February 10, 1934, J. H., a small Japanese boy of seven was carried into our office by his father. He complained of pain, placing his hand over the pit of his stomach, and asked for water. Vomiting and retching at intervals of three to four minutes, he brought up teaspoonful quantities of bile-stained mucus and saliva. He had been vomiting and retching almost continuously since 7:00 A.M., when he had cried out in bed with a severe pain in his abdomen.

The boy had been quite well and active until one week ago, Sunday, February 4, 1934. On that afternoon he had complained of some stomach ache, his parents at the time attributing it to over-indulgence in "apples and soda pop" at a picnic that day. Pain would come at intervals of thirty minutes to two hours followed by vomiting. The following day, with intermittent pain still present, he vomited each meal almost as soon as it was eaten. On Tuesday, the third day of illness, he was taken to a physician who made a diagnosis of appendicitis, nevertheless prescribed a mixture of Karo, orange juice, and water followed by a dose of castor oil! The patient had 3 soft bowel movements that night, apparently of normal color. His illness continued without any significant change until Friday night, the sixth day, when an enema was given, returning "dark blackish liquid matter."

On Saturday morning, the seventh day of illness, and the morning the patient was first seen, the parents stated that he cried out in bed with a sudden attack of pain that seemed more severe than usual, following which he began vomiting and retching almost continuously, pain coming at intervals of two to three minutes.

The family history was essentially negative. Both parents are alive and well, and four brothers and three sisters are likewise enjoying good health. There has been no familial history of tuberculosis or gastrointestinal diseases, and we were able to obtain no history of irregularity in the patient's eating or bowel habits.

Clinical Findings: Physical examination revealed a dark-skinned Japanese boy of seven, small for his age, lying on the examining couch with knees slightly flexed. His face was noticeably flushed, circum-oral pallor was present in contrast, and mucous membranes appeared slightly pale. He vomited and retched at two to three minute intervals. Oral temperature registered 99°F., pulse 155, thin and fleeting, respiration shallow, rate 25. He presented a general picture of collapse.

No injuries or lesions were evident on his skin or scalp. His head and sensory organs were negative, tonsils were small and uninfected. A few carious deciduous teeth were present, but none were ulcerated, chest examination revealed no abnormality of heart or lungs.

The abdomen was flat to the point of being scaphoid, and there was visible and palpable evidence of dehydration. Abdominal rigidity was very marked with a slight but distinguishable greater resistance in the epigastrium and the right lower quadrant. Tenderness to moderate pressure was present in the epigastrium near the navel, and in the right lower quadrant. No tumor masses were palpable, there was no visible peristalsis and no auscultable peristaltic rushes. Rectal examination, digital, was essentially negative.

Urine analysis was negative. The blood count revealed a leucocytosis, 17,000, with 90 per cent polymorphonuclear neutrophiles.

Surgery: February 10, 1934, 5:50–6:35 P.M.

Preoperative diagnosis: Acute surgical abdomen, possible ruptured appendix with general peritonitis.

Postoperative diagnosis: Duodenal ulcer, acute perforation.

The abdomen was opened first through a low right lateral rectus incision. Immediately upon section of the peritoneum a moderate amount of bile-colored fluid with occasional bubbles of air welled up into the incision. The lower ileum

and cecum were quite distended by gas, tending to balloon up through the wound, within the bowel lumen blackish lumps of clotted blood were visible. The appendix was immediately found in the anterior position, uninflamed; it was removed in the usual manner. Because of the presence of bile, the incision was enlarged upward to the costal margin. The liver and gall bladder were seen to be normal. On approaching the gastroduodenal area flow of bile and bubbles was noticeably increased, on the ventral surface of the duodenum about 1.5 cm. from the pyloric ring was found a round, symmetrical, clean-cut perforation about 0.5 cm. in diameter. Bile was issuing from this perforation in a small rhythmic stream, bubbles forming at the outlet. There was no marked inflammatory reaction about the perforated ulcer and no evidence of any chronic inflammatory process, the area immediately surrounding the ulcer being soft and relatively normal in color. The perforation was closed by a purse-string suture, the closure then sealed over with a row of Lembert sutures, and fortified with a small tag of omentum. Further examination revealed no other abnormality of stomach or duodenum. The abdominal cavity was then irrigated with 3 liters of hot normal saline, no food particles were found. Closure was effected in the usual manner, one Penrose drain was placed to drain the right lumbar gutter and one the lower abdomen. After a transfusion of 200 c.c. of donor's blood the patient was transferred from surgery in good condition. The postoperative course was uneventful, and the patient was discharged from the hospital on the fourteenth day.

DISCUSSION

The general impression gained from a review of the literature is that duodenal ulcer in infants and children is, unfortunately, largely a postmortem diagnosis. Such a status would necessarily obtain when records reveal that the greater number of cases occur in infancy when clinical diagnosis labors under a severe handicap. Holt[2] pointed out that in the 95 infant cases which he studied from the literature no symptoms suggestive of the condition were present in over one-third of them, the ulcer being discovered at autopsy in patients dying from other causes. Ulcers in infancy are usually acute in nature, and hemorrhage is the only definite symptom pointing to duodenal ulcer; blood, either free or chemically changed, may be vomited or passed in the stool. Experience has shown that the marasmic infant, and the "melena neonatorum" should be regarded with suspicion.

In view of the present uncertainty of diagnosis surgical procedures in infants would seem rarely justifiable, yet, if the case is seen and diagnosed early enough, surgical treatment—simple closure, pyloroplasty, or closure of the ulcer with gastroenterostomy—is not an unreasonable procedure. Infants stand the loss of blood very poorly, and in the face of continued hemorrhage expectant medical treatment is an unsafe policy. Palmer[12] successfully performed a posterior gastroenterostomy in a child of six months with duodenal ulcer, after an initial pyloroplasty had failed.

Henderson[13] is led to the interesting conclusion that many of our so called "difficult feeding" cases are probably ulcers, many under-developed, under-nourished children could have been cured and fattened on an ulcer regime. It is quite probable, too, as Bloch et al[8] have observed, that many ulcers in children are not diagnosed because gastrointestinal upsets during childhood are given such slight consideration. Many of these ulcers undiagnosed in childhood are not discovered until they enter the realm of adult medicine. At the Mayo Clinic Proctor[5] discovered that out of 1000 gastric ulcers 16 of the patients had symptoms dating back to childhood, even to the age of four or five years. Likewise in 1000 cases of duodenal ulcer 26 patients in every 1000 afflicted with chronic peptic ulcer had the onset of disease in childhood.

In children above the age of one, history and the usual clinical and laboratory procedures offer a reasonable chance for diagnosis. It is realized, however, that the ulcer problem in children is rendered extremely difficult by the frequent paucity

or complete absence of symptoms. Such "silent ulcers," nevertheless, are not infrequently discovered in adults by the observant clinician. In a classical manner Proctor[5] has so clearly stated that "The most important single factor in the diagnosis is the realization that chronic peptic ulcer occurs in children."

Though it may be absent in the earlier years the familiar pain syndrome becomes more and more a clinical entity as the age of incidence approaches the age of puberty. Dickey[7] found that epigastric pain in some form is a common, if not the most common sympton and complaint. Mid-epigastric tenderness is usually elicitable. Hunger pain with ease on ingestion of food has also been a usual finding, while nausea and vomiting are present in most cases. Proctor,[5] Dickey,[7] and Henderson[13] concur that nocturnal pain is a very frequent complaint. At times the signs and symptoms may be so diversified that no diagnostic value can be placed, thus Kennedy[14] urges that duodenal ulcer be sought for in all children who have chronic or recurring indefinite abdominal pain. It has been well proved that radiologic study is the most valuable single procedure in diagnosis.

The treatment of chronic peptic ulcer in children is essentially the same in principle as the treatment in adults, modifications being made when necessary. Medical treatment should be the treatment of choice unless conditions are present which frankly contraindicate it as in adults: repeated hemorrhages, perforation or stenosis. Uniform success with bed rest and a modified Sippy diet has been reported by Dickey[7] in his 3 cases, and of the 5 cases reported by Bloch, Bronstein, and Serby[8] it failed in only one, in which there was an associated allergic condition.

Surgical treatment is indicated in the event of perforation, repeated or continued hemorrhage, or failure of medical treatment. The regenerative and reparative powers of children lend well to early recovery by either medical or surgical treatment as the case may be. Proctor[5] of the Mayo Clinic concludes that the simplest operations are the best, advocating for the duodenal ulcer a simple excision, or excision and pyloroplasty after the method of Judd or C. H. Mayo. In the event of marked pyloric stenosis gastroenterostomy or even partial resection may be necessary.

COMMENT

A case of perforated duodenal ulcer in a seven year old boy has been presented. It differs essentially from other recorded cases in that the perforation occurred in an acute ulcer giving no history of gastrointestinal disorders extending beyond seven days. There was no history of trauma, or cutaneous lesion, and no focus of infection was present outside of a few carious deciduous teeth.

The case reported by O'Flyn[9] is of particular interest because of its many similarities to the present case.

The patient, a boy of fourteen, had been taken violently ill the previous evening with severe abdominal pain. Put to bed the patient spent a restless night with vomiting and considerable distress. Being unimproved the following morning, the doctor was called. "When first I saw him, he looked very fit and did not complain much; his temperature was 102°F., and his pulse 140; he was generally tender all over the abdomen with perhaps a particular selection for the region of the right iliac fossa. The tongue was muddy and offensive. I determined he had a perforated appendix with general peritonitis, and at once ordered his removal to a hospital." At operation the abdomen was opened through a right lateral rectus appendectomy incision. The appendix was found normal and removed. Extending the wound upward to expose the stomach, an ulcer was found in the anterior wall of the first part of the duodenum. "The ulcer was about the size of a shilling, hard and crateriform and had at its base a perforation about the size of a pea." Closure of the perforation was effected with a fold of gastrohepatic omentum and posterior gastroenterostomy was performed. The patient made an uneventful recovery and went home in sixteen days.

The ulcer in O'Flyn's case was chronic, but it is interesting to note especially the similarity of preoperative diagnosis "perforated appendix with general peritonitis." This leads one to conjecture how many such diagnoses may have been made in the past, the patient expiring later after removal of an innocent appendix.

Our patient has been seen on several occasions during the past six months, he has had no complaints at any time and at present he is enjoying an active healthy life. On August 7, 1934, practically six months from the time of operation a gastrointestinal *x*-ray study was done merely out of curiosity, a verbatim report of which is submitted below.

Radiographic and fluoroscopic observation of the stomach shows a stomach of medium to large size, active tone, hypertonic type, axis vertical. The position of the stomach is high, the lower pole being above the level of the anterior superior spines of the ilium. The pylorus and bulb are situated to the left of the median line level with the body of the third lumbar vertebra. The stomach as a whole is freely movable.

Antral activity and evacuation is prompt and active. Peristalsis is very active. There is no persistent defect in gastric outline, but the duodenal cap is never well filled and is tender on palpation. At six hours there is a large residue in the stomach in the pars media and pars pylorica. The barium shows an intestinal hypermotility in the cecum, ascending, transverse, and descending colon.

Diagnosis: Obstructive lesion near the pylorus. Possible duodenal or surgical adhesions. S. V. Kibby, M.D.

SUMMARY AND CONCLUSIONS

1. Gastric and duodenal ulcers in infants and children, though rare, do occur, probably with greater frequency than is at present admitted.

2. The most important single factor in diagnosis is recognition of the fact that they do exist.

3. Duodenal ulcer should be sought in children giving a history of chronic or recurrent vague abdominal pains.

4. Radiologic study is the most valuable single procedure in diagnosis.

5. Granting an early diagnosis, in the face of continued hemorrhage surgical treatment is feasible even in infants.

6. Medical treatment is the treatment of choice, surgical indications being essentially the same as for adults.

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PEZZER CATHETER GASTROSTOMY

ALEXANDER BRUNSCHWIG, M.D., F.A.C.S.

Department of Surgery and Division of Roentgenology, University of Chicago Clinics

CHICAGO

IN view of the several exhaustive treatises on operative technique, in which several procedures are usually described for the attack of almost every situation that the surgeon may encounter, any further brief report in this field must appear very trite. However, in regard to gastrostomy, there has seemed, to the writer at least, room for some modification of the operations as usually recommended. Without a lengthy discussion, it may be stated that the perquisites of a satisfactory gastrostomy include: (1) Simplicity of technique to permit rapid execution, since patients subjected to this procedure are usually in poor condition. (2) The tube should be easily retained *without recourse to suture of it to the stomach or abdominal wall.* (3) There should be no leakage of gastric contents about the tube. (4) It should be possible to remove the tube or to change it very easily.

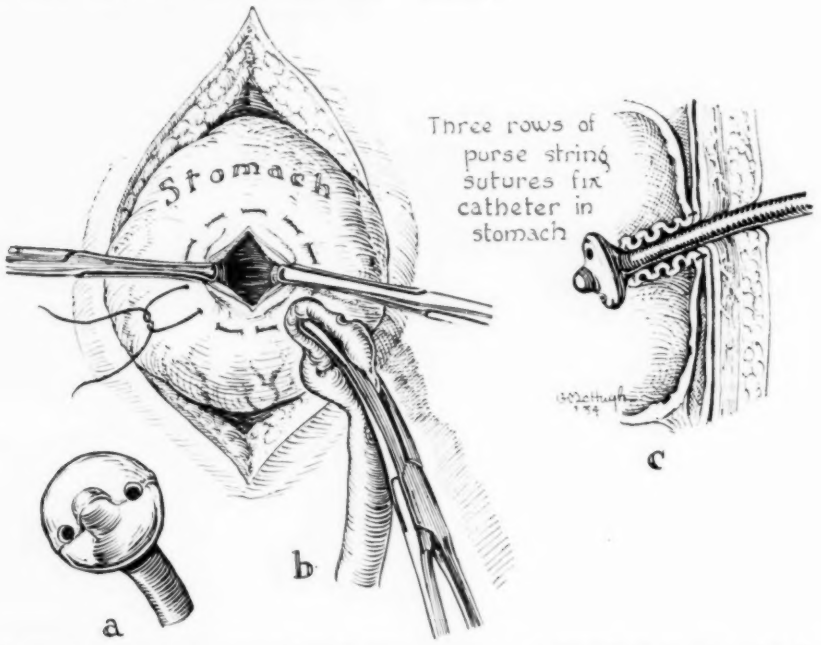

FIG. 1. Essential features in gastrostomy using Pezzer catheter as described in text. (a) Large phalange of Pezzer catheter, size 26 F. (b) Insertion of catheter into stomach. (c) Diagrammatic representation of catheter in place after completion of operation.

In the palliative treatment of carcinoma of the esophagus and carcinoma of the upper portion of the stomach with extension into the esophagus, the writer performs a gastrostomy followed by intensive 200 kv. *x*-ray therapy to the lesion. During the period of irradiation and following it the patients are nourished entirely per gastrostomy; nothing is permitted by mouth. If, following the subsidence of the skin reaction from irradiation, the patient can gradually return to nourishing himself entirely by mouth, the gastrostomy tube is withdrawn.

The type of gastrostomy that has proved satisfactory in this program is as follows:

Step 1. A high midline incision is made under local anesthesia. If exploration is to be done, it is, of course, made large enough to admit the hand. Otherwise it need not be more than 8 cm. in length.

Step 2. The lower anterior wall of the stomach is brought out through the incision by traction with Allis forceps.

Step 3. At a point midway between greater and lesser curvatures, a purse-string suture of fine silk is inserted about the site for perforation. The stomach wall is then perforated. The phalange of the Pezzer catheter is compressed by a large curved Kelly forceps and is inserted into the stomach, the forceps withdrawn and the purse-string suture tied, firmly but not very tightly, about the catheter. The stomach wall is then further invaginated about the catheter by two more successive purse-string sutures of silk.

Step 4. A stab wound, a little over 1 cm. in length, is made through the abdominal wall, 3 cm. to the left of the midline incision. The free end of Pezzer catheter is then brought through the stab wound.

Step 5. The stomach wall about the catheter is sutured to the parietal peritoneum by interrupted silk sutures. When possible omentum is included with these sutures.

The midline incision is closed.

Strips of sterile gauze are tied about the catheter and pressed firmly down upon the skin.

The operation described is a modification of the Stamm or Senn gastrostomy, in which an ordinary urethral catheter is used, requiring its suture to the abdominal or stomach wall for retention, and of the Dragstedt gastrostomy in which a metal cannula is used. The Dragstedt operation is essentially a permanent gastrostomy, since a second laparotomy is necessary to remove the cannula.

The Pezzer catheter gastrostomy has been performed twelve times. The longest period in which a patient was completely or partially nourished by gastrostomy was seven months. The original tube was employed during this period. It was not necessary to remove it for cleansing or

Fig. 2. Photograph taken seven weeks after Pezzer catheter gastrostomy; there is no leakage of gastric juice and skin shows only a slight erythema about catheter. Area of maximum epithelitis due to intense 200 kv. *x*-ray therapy administered for carcinoma of cardiac portion of stomach with extension onto esophagus.

because of any damage by gastric juice or wound secretions.

Leakage of gastric contents about the tube occurred in only one instance, where it began five months after operation and was due to growth about the gastrostomy of a large carcinoma of the upper half of the stomach involving the lower esophagus.

There was one postoperative death (seventh day), also in a case of carcinoma of the stomach involving the esophagus. Although no post-mortem was performed, the clinical diagnosis was peritonitis, probably due to soiling at the operation. Both midline incisions and stab wound remained clean and there was no evidence of leakage about the tube.

In no instance did the tube fall out spontaneously. In no instance was there appreciable infection in the stab wound or digestion of the skin about it. A small

amount of mucopurulent discharge occurs about the tube, which necessitates daily change of dressing. (This is done by the patient.) Exuberant granulations may develop about the catheter necessitating cauterization by silver nitrate.

The tube may be removed by progressive firm traction or by first inserting an obturator to elongate the phalange. It may be reinserted with the aid of a Pezzer catheter obturator. During the period in which the tube is out and the patient is nourishing himself by mouth the gastrostomy sinus may be kept open by insertion of a short solid rubber bougie. This precaution facilitates reinsertion of a catheter if and when obstruction to swallowing by mouth recurs.

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REACTION OF THE PERITONEUM

AS IT AFFECTS THE SURGICAL PATHOLOGY OF PERITONITIS*

J. W. KENNEDY, M.D.

Surgeon in Charge, Joseph Price Memorial Hospital

PHILADELPHIA, PA.

IT is a legitimate tendency for one to combat the teaching of our profession in any condition where privilege and execution differ in a marked degree.

During the past three or four years hundreds of articles have been written on the increasing death rate from the peritonitis principally due to the ruptured appendix.

If this gross amount of literature is reviewed, it will be found that there is nothing advocated that is new. Indeed, there is such a marked accord in the common teaching of the day, that we are not stimulated by new thoughts and therefore have little to look forward to as progress.

In other words, the teaching of the profession of today in the peritonitic abdomen is of common adoption, quite standardized and the death rate has been rapidly increasing during the past twenty odd years.

GENERAL DISCUSSION

My discussion of this subject is an appeal from the popular teaching and advice, surgical or medical, in the case of the peritonitic patient.

We differ in nearly every particular in what is common teaching in the care of the patient who is gravely ill from peritonitis.

As at least ninety-six or ninety-seven per cent of the literature concerning the peritonitic patient is a discussion of the condition emanating from a ruptured appendix, we will confine our discussion in a large measure to this source, only touching on other types of peritonitis.

BIRTH OF THE PHYSIOLOGIC AGE IN SURGERY

Twenty odd years ago a number of eminent physiologists and pathologists of Europe brought forth the function of the white blood cell as a phagocyte in its protective function in combating infection.

This teaching was seized upon by a number of American surgeons, who adopted the "hands off" method of treating peritonitis with the idea that the phagocytes should be permitted to function undisturbed. Thus, the so-called physiological age in surgery was born.

In 1910 in a paper reviewing the working factors of the physiological surgeon, we made the prophecy that the death rate in appendicitis and its complicating peritonitis would double itself. It has done so in many communities. Indeed, the death rate in appendicitis is not properly recorded, as many deaths are registered as due to the complications of the conditions, such as bowel obstruction, abscess of the bowel, postoperative pneumonia, etc. This is all wrong; the appendix was the primary cause of the condition and must be held culpable for all complications causing death.

The death rate from peritonitis following the ruptured appendix has always been high; indeed, it is a nightmare to the surgeon or physician, so it was an easy matter to turn the operating profession from what had been more or less popular teaching to the endorsement of a different view.

Especially was this so, as the working factors of the physiological surgeon were

* Read before the American Association of Obstetricians, Gynecologists and Abdominal Surgeons.

easy of accomplishment. The "hands off" method could be accomplished by the tyro. Nature will have her toll; a thing half done must be done again and this is what has followed the indifferent surgery of the day in dealing with the peritonitic patient. Continued infection, multiple operations, numerous postoperative bowel obstructions, distal abscesses are telling evidences of inferior primary work.

In order that we may more intelligently make a discussion of the reactions of the peritoneum, it is necessary that we review some of the views of the physiological surgeon.

WORKING FACTORS OF THE PHYSIOLOGICAL SURGEON

It may be said that the entire plan of the surgical procedure or thought of the physiological surgeon is to prevent absorption of toxins by the peritoneum; no other source of infection is to be considered. The entire therapy of the conditions stands on a single leg.

Therefore, the following steps were endorsed by the physiological surgeon:

If the patient is seen in a peritonitic stage, no operation is to be done; the patient is put in the Fowler position and the watchful waiting plan is endorsed until there is a quiescence of the active symptoms. Saline and glucose may be given in any amount. If the patient is seen when there is evidence of a local abscess, an incision may be made, the appendix removed if easily accessible, but under no circumstances must the appendix be removed, if adhesions are to be broken in order to reach the offending organ. The adhesion is looked upon as sacred and must not be broken on account of fear of spreading the infection.

The patient with general tenderness and distention is recognized as one with diffuse peritonitis and active surgery in every particular is contraindicated.

Therefore, all possible complications of the peritonitis are ignored and but one possible source of infection is recognized, namely, that which emanates from absorption of toxins from the peritoneum. In other words, the peritonitis per se kills the patient.

REACTIONS OF THE PERITONEUM

This brings us in our discussion to the reactions of the peritoneum. We take the position that reactions of the peritoneum from infection are defensive rather than offensive; that the peritoneum seems to have almost a human sagacity; it will absorb kindly fluid, such as saline, but rejects irritating and infectious fluids by throwing out protective fluids and lymph barriers in an effort to prevent absorption.

FUNCTIONS OF THE PERITONEUM

We take the position that the existing peritonitis is in a sense physiological and were it not for the fact that the peritonitis superimposes upon itself bowel obstructions, distal abscesses, etc., the peritonitis per se would quite uniformly win the fight.

With this view in mind, we teach in the surgical management of the case, that it is not the peritonitis which will give the final and fatal dose of infection, but it is the complications of the peritonitis which must receive urgent surgical consideration.

In other words, we operate for the complications of the peritonitis and not the peritonitis as a pathologic entity. With this thought, our surgical executions necessitate evisceration of the peritonitic organs, which, of course, is a vile intrusion upon all that is taught by today's popular endorsement.

We further teach that the peritonitic membrane absorbs little and that the same can be surgically manipulated with little fear of increasing absorption of infection and that the peritonitic structures may be properly dealt with with less fear of shock than the normal or physiological structures.

We have proved this to our full satisfaction, as we eviscerate each peritonitic patient and have not seen a case of typical secondary shock.

During the past twenty-five years, there is not a record of a death from typical shock in the Joseph Price Hospital, except

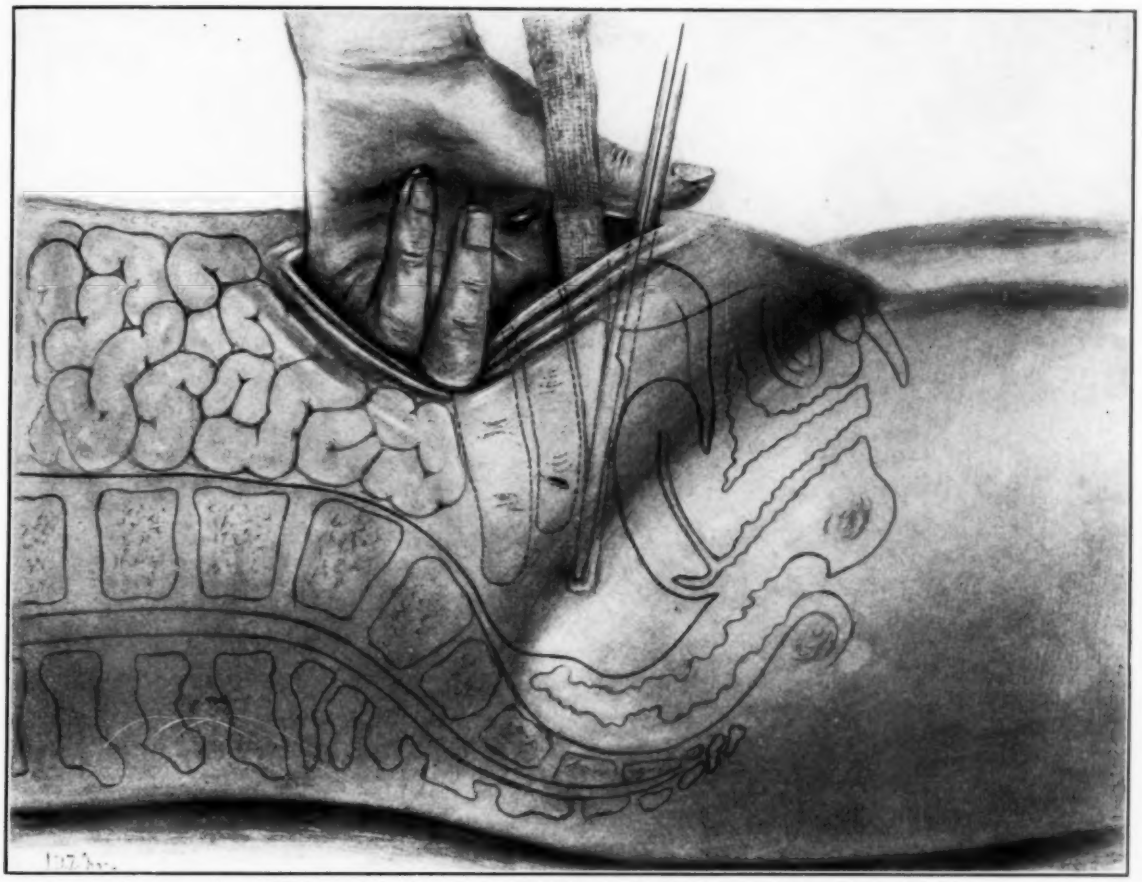

Fig. 1. Beginning insertion of Price coffer-dam. In general discussion point was brought up that ere any attempt was made to use Price coffer-dam it was necessary to have removed offending pathology. Figure shows two big fingers of left hand have been inserted quite to bottom of pelvis and back of hand with aid of these two big fingers is controlling viscera in order to prevent them from collapsing into pelvis.

A long dressing forceps is grasping a piece of gauze and is passing it to most dependent point of pelvis and this is continued, piece against piece, until entire pelvis is filled, as next figure well shows; same principle of inserting coffer-dam is carried out in any other part of abdominal cavity. It must always be kept in mind that coffer-dam is a continuous solid wall, never two or more columns, as has been brought out in general discussion. (From Kennedy's "Practical Surgery of the Abdominal and Pelvic Regions." F. A. Davis & Co.)

those cases in which there was no evidence of intra-abdominal infection.

This is probably due to the fact that the normal peritoneum is more receptive to trauma and absorption than a pathologic structure, which already has its receptive centers intoxicated or insensible to depressing factors of trauma or infection.

Function of the Lymphatic Vessels

Our experience with the peritonitic abdomen does not permit us to accept the teaching that the lymphatics are the true absorbents of the peritoneum, nor do we feel that they have their beginning in the peritoneum. It is our opinion that the blood vessels are the true absorbents of the peritoneum.

Doctor Hertzler points out the fact that if a dye is placed within the peritoneal cavity, the dye will appear in the urine before it is seen in the lymphatics, showing that the stain has gone through the general circulation before it appears in the lymphatics. Therefore, the lymphatics are not true absorbents; they belong probably to the sewer system of our anatomy. The amount of lymph produced does not depend upon absorption of fluids, but

comes from metabolic function and depends largely upon such source. This is in accord with our teaching.

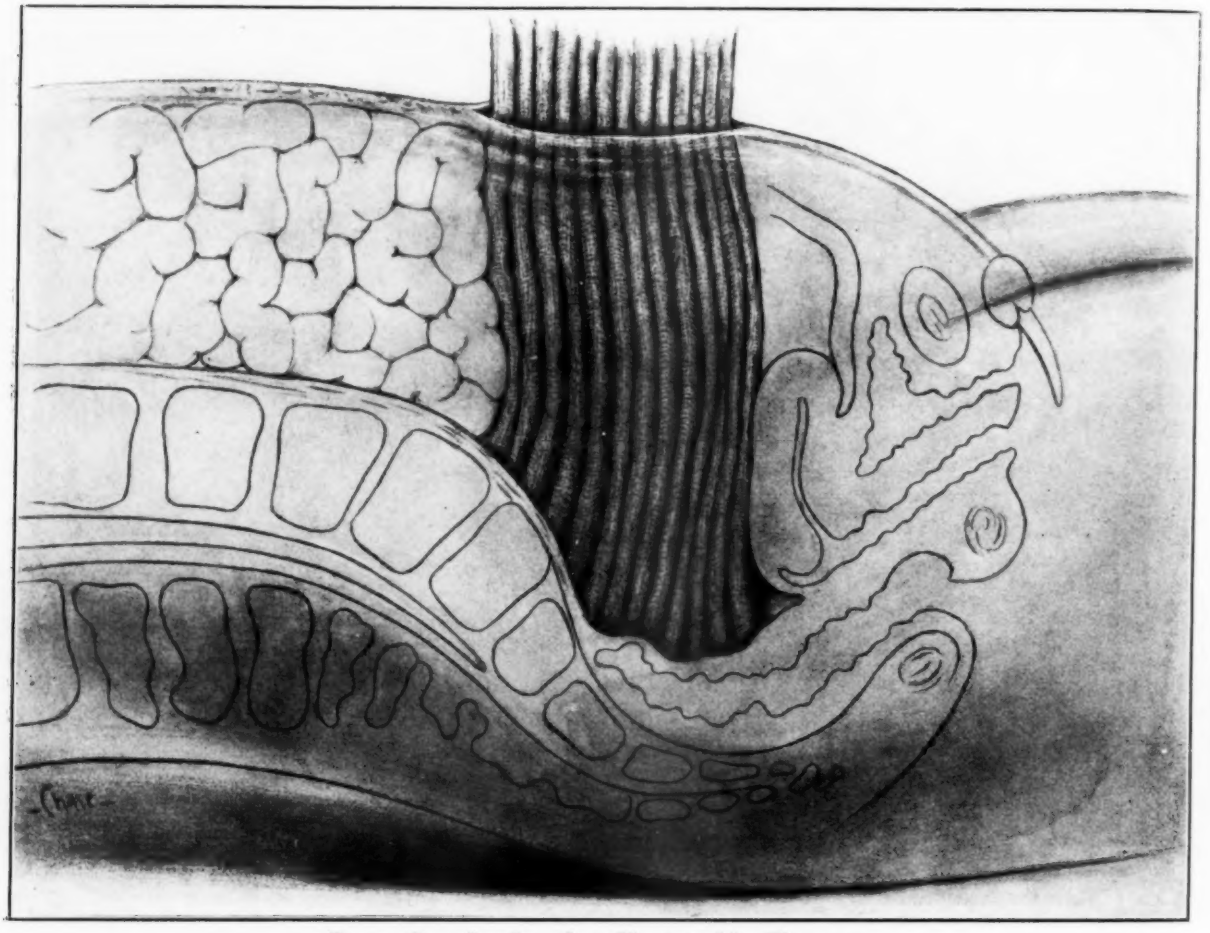

Fig. 2. Completed work as illustrated by Figure 1.

Coffer-dam is a solid wall composed of an indefinite number of pieces of plain gauze 1 inch wide folded so as to make six thicknesses which is a convenient size for easy insertion. If these strips of gauze are too big they are unwieldy and there is excessive trauma in their insertion.

These pieces of gauze are inserted as figure indicates, side by side, just as piles are driven. This coffer-dam is never in any sense a pack, as such insertion would be apt to crowd down the viscera and produce a postoperative bowel obstruction. It can be seen how viscera are held from pelvis or any dependent filthy area to which coffer-dam might be inserted. If infection extended from pelvis up into appendiceal region coffer-dam would be continued to that point which marks extent of infected area. (From Kennedy's "Practical Surgery of the Abdominal and Pelvic Regions." F. A. Davis & Co.)

Doctor Hertzler further points out there is no greater example of reasoning on a false premise than to assume that any one area of the peritoneum is richer in absorbents than any other.

This sustains the opinion that we have always had and for this reason we have never endorsed the Fowler position. We must give up the idea that the stomata throughout the peritoneum are the beginnings of the lymphatic vessels and are wide open mouths ready to absorb intra-abdominal fluids.

We have discussed at some length and illustrated in a monograph entitled "Practical Surgery of the Abdominal and Pelvic Regions" the position in which we place the peritonitic patient after operation and the method of dealing with adhesions. Some of these illustrations are shown in this article.

Any operator who has eviscerated the peritonitic organs is constantly seeing evidence of how very little gravity has to do with drainage in any peritonitic condition.

The peritonitic cavity should be looked upon as a huge carbuncle with its numerous partitions that quite separate areas which may be enclosing distal abscesses and which, after all, are often the cause of the complete or partial bowel obstruction and such abscesses remain undrained by any position.

The physiological surgeon has not learned this. We feel that the discussion of the surgical pathology of peritonitis will have to be rewritten entirely. We must not look upon the peritoneum as a great sheet of tissue, nearly the area of the skin of our entire anatomy, with the idea that it is just a finishing layer covering the abdominal organs and lining the inner-abdominal wall. The peritoneum has little suspensory function and must not be surgically used for that purpose.

A much better mental picture of the function of the peritoneum will be obtained

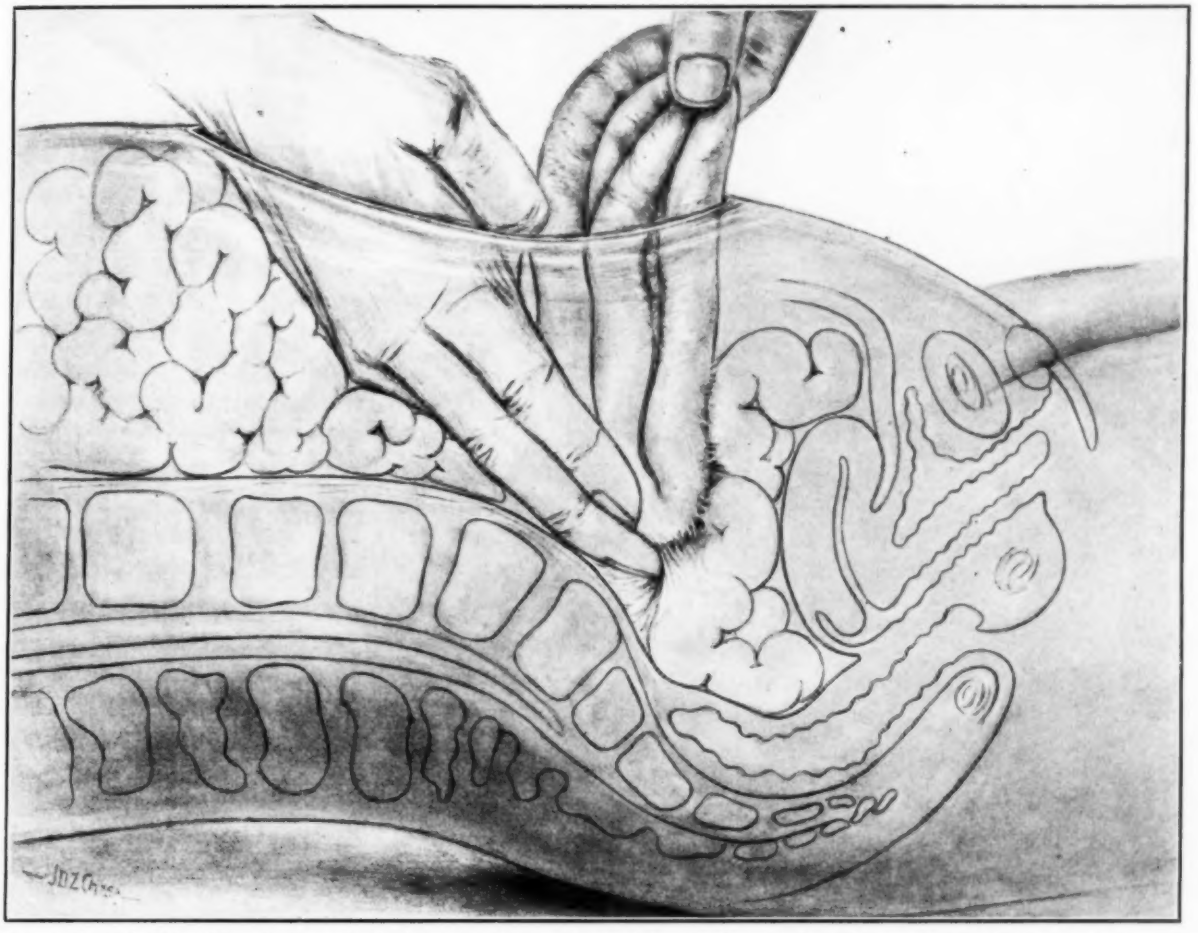

Fig. 3. Joseph Price Hospital method of attempting to deal with an adherent bowel or with any fixation of viscera. We tried to bring out in our general discussion in a number of places that adhesions must never be broken by traction on viscera, and by this figure we are attempting to demonstrate release of a knuckle of bowel from its fixed point.

It can be seen that operator is making slight traction on bowel by right hand while two big fingers of left hand are making an attempt, so to speak, to lift bowel from its anchored point. We try to insinuate fingers beneath fixed point of bowel, making pressure at expense of adhesions and not viscera. This is, of course, another way of saying we are seeking a line of cleavage between viscera, but even more gentle work than mere enucleation of a specimen must be done in dealing with firmly fixed bowel.

Ability to deal with fixed small intestine has much to do with peritonitic work. Great care should be taken not to make more traction on fixed bowel than will enable one to define its fixed point, release of such fixed point is done, as we have said, by attempting to lift bowel from its anchorage by insinuating finger beneath it. (From Kennedy's "Practical Surgery of the Abdominal and Pelvic Regions." F. A. Davis & Co.)

when the medical profession is taught that the peritoneum has a physiological function per se just as the kidney or liver has and that its reactions are quite uniformly defensive rather than offensive.

It is a blessing for us who do surgery that this is true, as we are all clowns in our surgical executions. We do not contend that infectious fluids placed within the abdominal cavity will not kill the patient. Such fluids will pass through the peritoneum and therefore might be classified as a death due to peritonitis. This may be seen in the frank perforations of the abdominal organs, such as a perforation of the stomach or duodenum or pos-

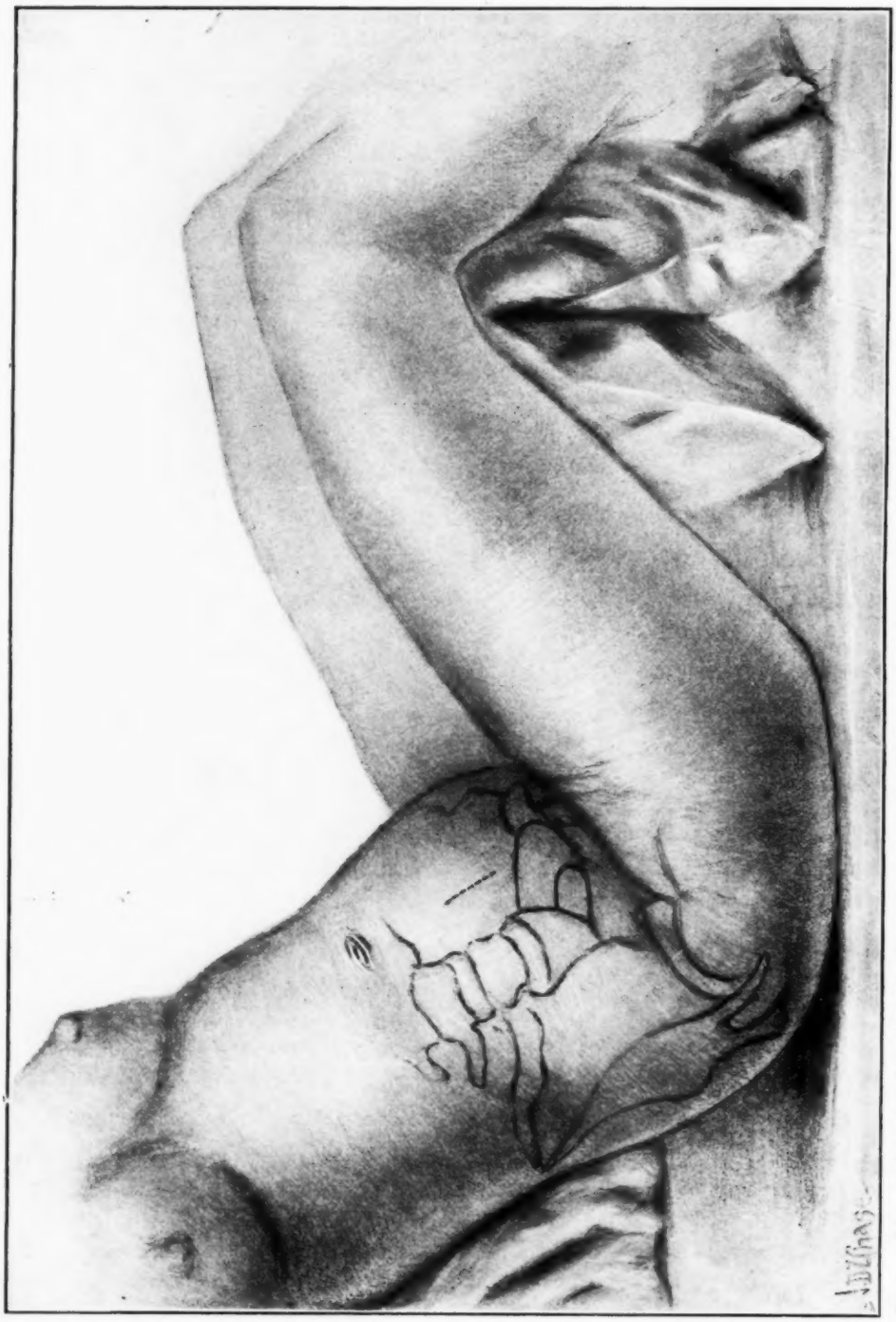

FIG. 4. If a patient is placed in Fowler position and incision is made at point indicated by dotted line above pubes, pelvis will be a basin 6 or 7 inches deep and will contain infected fluids in peritonitic patient. It was our contention brought out in general discussion that patient in Fowler position was poorly drained and that surgeon did not in reality obtain the most dependent point for drainage as he had anticipated from use of Fowler position.

It is our contention that by an incision made at McBurney's point and patient turned on right side, such lateral position was in reality a better postural one for drainage. Student by making a tudy of this figure and figure 5 will be able to see just how pelvis will empty itself when patient is in right lateral position and also how pelvis will retain a great quantity of fluid when sitting in Fowler position with a suprapubic incision. (From Kennedy's "Practical Surgery of the Abdominal and Pelvic Regions." F. A. Davis & Co.)

sibly in a sudden perforation of the very large and distended appendix filled with black foul fluid, where there has been no effort by nature to prepare the peritoneum for such an overwhelming dose of infection.

Many of these patients will die before there is any evidence of a marked peritonitis. These cases are brilliant examples of deaths due to the absence of a peritonitis rather than from such source of infection. The toxins had passed through the peritoneum with little or no reaction from the membrane and had become rapidly a blood vessel infection or septicemia and cannot be classed as fatal termination from the peritonitis.

Any operator who eviscerates the peritonitic organs will learn that the cases

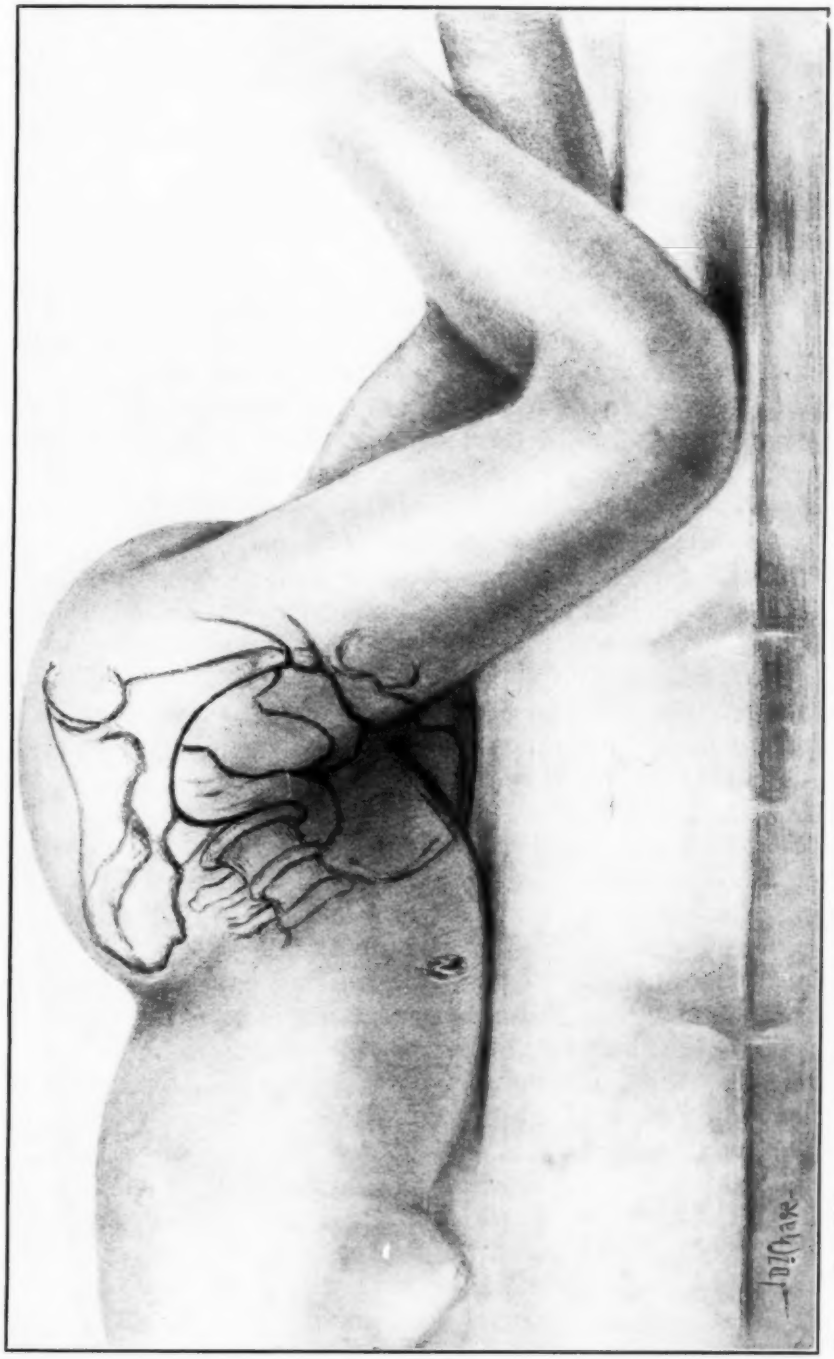

Fig. 5. It will be seen that if patient is turned well over on right side with incision at McBurney's point or at location indicated in this figure by dotted line, pelvis would quickly empty any free fluids it might contain. This is even more true if patient's right hip is pulled well back toward edge of bed, patient is thrown in a position midway between a right-lateral and prone position, which even better empties pelvis. If there is anything in postural drainage it will be obtained by placing patient in this position immediately after operation.

It must be remembered if patient is in Fowler position and has been operated for a peritonitis that any drainage tube placed in pelvis is minor and inferior drainage and is quickly localized. We brought out in discussion that influence of postural drainage in peritonitic abdomen saw much exaggerated as fluids did not gravitate freely through barriers incident to a peritonitic abdomen. Our object in showing Figures 4 and 5 is for student to see for himself that Fowler position does not give most dependent point for drainage, which in such position is far below incision into abdominal cavity and will thus retain fluids. (From Kennedy's "Practical Surgery of the Abdominal and Pelvic Regions." F. A. Davis & Co.)

which show the most marked evidence of peritonitis will not have as high mortality as the cases where there is little evidence of peritonitis, but marked indications of a deep retroperitoneal infection.

This condition is referred to by us as a dry peritonitis because there has been little exudate thrown out by the reactions of the peritoneum. This type of abdominal infection is seen in the perforation of the retroperitoneal or sub-cecal appendix and it is also the type of all puerperal infections, which should not be classified as examples of a true peritonitis.

These infections start as retroperitoneal involvements and continue and extend as such. We must have more accurate teaching of the pathology and surgical pathology of the puerperal infections.

Although a peritonitis may be evidenced in a patient who has died from a puerperal infection, however, the reactions of the peritoneum manifested as a peritonitis were principally caused from a retroperitoneal source and not primarily as reactions of the peritoneal membrane.

If a fatal termination follows this type of retroperitoneal infection from any source, it will be seen that the patient died with a flat abdomen and soluble bowel, which is never seen in a fatal termination from a typical peritonitis.

SOURCES OF PUERPERAL INFECTIONS

We take the position that the final and fatal dose of toxins in the puerperal patient comes from the retroperitoneal infection and that the peritoneum becomes involved secondarily.

All puerperal infections are wound infections primarily and spread or travel in continuity of structures, blood vessels, etc., unresisted and unlimited between the two great resisting planes of our anatomy; namely, the mucous membrane of the birth canal and the peritoneum. The infection in the birth canal is submucous; as it extends to the abdominal cavity, it continues as an unresisted retroperitoneal infection. For this reason in performing a cesarean section in the infected patient, I am much more in dread of cutting into the infected uterine structures than I am fearful of a fatal termination from a peritonitis.

CRAMPED VIEWS OF THE PHYSIOLOGICAL SURGEON

The views of the physiological surgeon in the peritonitic patient remain in status quo. He is not permitted to eviscerate the abdominal structures and thus familiarize himself with the complications of the peritonitis.

With such a cramped toilet he, from necessity, remains ignorant of the extent of involvement of the peritoneum. This is evidenced in every particular in his discussions of the surgical pathology of the condition.

The treatment of the peritonitic abdomen is drainage in its broadest sense and the feeble efforts at drainage by the physiological surgeon exposes his knowledge of the extent of involvement and the superimposed complications of the condition.

We do not hear the physiological surgeon discuss 2, 4, 6, or 8 feet of peritonitic bowel, yet he speaks of a diffuse peritonitis and reports recoveries from the same.

It is absolutely necessary, before the present-day teacher can compare mortalities in these peritonitic conditions with the operator who eviscerates the patient, that the physiological surgeon demonstrate the extent of the lesion.

A great number of times I have called attention to the fact that if the last 6 or 8 inches of the ileum are peritonitic from a ruptured appendix, there will be found often general distention and tenderness due to this small extent of peritonitic and paralyzed bowel. This case would be reported by the physiological surgeon as a recovery from a diffuse peritonitis, whereas there had been but 6 inches of small bowel involved.

Again, the physiological surgeon who has endorsed the watchful waiting for

subsidence of acute symptoms must report all deaths which occur (and there are many) during the watchful waiting period, although no surgery has been done.

These surgical cards must be laid upon the table before results can be compared with the operator who operates the first hour, irrespective of the stage of the peritonitis.

TEACHING OF TWO SCHOOLS

What hopeful progress may be seen in the teaching of the two schools?

The surgeon who operates the first hour he sees the patient and exposes the extent of the peritonitic involvement, severely rebukes the tardy physician through an exposition of the peritonitic structures and drives any thinking physician toward early operative hour. He has exposed the tardy physicians' mistake.

How about the watchful waiting plan of today's popular teaching?

The patient comes to the hospital on Wednesday, the physician in charge recognizes a supposed diffuse peritonitis in the acute stage and places the patient on the watchful waiting list for subsidence of acute symptoms, possibly telling the attending physician that had he seen the patient on Sunday or Monday he might have operated, but that at present the stage is too advanced for surgery.

The family physician leaves with a confused idea and probably will wait next time for subsidence of acute symptoms ere he brings the patient to the hospital. I have a great number of times seen this and today physicians phone me that they have been watching a patient for a week and that at present the peritonitic condition has become a local abscess and therefore ready for surgery.

The discussion of this subject is endless; the crux of the situation is this: the teaching is what it is and of popular adoption as already indicated and the results are reprehensible with an ever-increasing mortality.

MORTALITIES

Accepting the classification of the physiological surgeon of the peritonitic patient, although he has not in any case demonstrated the extent of the pathologic involvement and although he does not count deaths due to watchful waiting, a mortality of 28.4 to as high as 40 per cent is given as the death rate in diffuse peritonitis from the ruptured appendix.

The mortality in peritonitis from the ruptured appendix in the Joseph Price Hospital has remained between 5 and 6 per cent, all cases being operated upon the first hour seen, all structures eviscerated and drained by the extensive system of coffer-dam of gauze. A large percentage of our mortality comes from the moribund case or the large fleshy patient with well-recognized cardiorenal symptoms, no patient being refused operation.

TREATMENT OF THE PERITONITIC PATIENT

We repeat that the treatment of peritonitis is drainage in its broadest sense and that we operate for the complications of the peritonitis and not the peritonitis per se.

In our surgery of the condition we begin with a mental picture of the complications; namely, partial and complete bowel obstruction, distal abscess, etc. In order to reach these complications, evisceration is necessary.

During the operation on these cases, we content ourself with any pathologic structure which first presents to our manipulation, be it omentum, small or large bowel, and continue the evisceration until the final source of infection is reached, be it the appendix, pus tube, perforated gall bladder, ruptured diverticulum of Meckel; we travel on continuity of pathologic structure until the source is reached and removed or surgically treated.

During this manipulation of pathologic structures, do not dive or delve into the peritonitic viscera in an eager attempt to reach the source of infection, but treat each

pathologic structure as it is eviscerated, realizing that such course of evisceration will bring the operator in the end to the primary source of infection. This is most important.

The peritonitis travels as the prairie fire, gradually spreading over continuity of structure, and does not jump over clean structures and produce distal abscesses in dependent points of the anatomy. For this reason we condemn the use of two or three drains inserted to dependent points of the anatomy with the idea that pus is in these dependent areas or will be there.

The surgeon who eviscerates the peritonitic patient knows that this is never so. To anticipate that any dependent point in the abdominal cavity contains infectious fluids and pass any kind of a drain through a clean area to such supposed infected dependent point, is to carry the infection to a clean area of the peritoneum.

The physiological surgeon never learns this. The operator who eviscerates knows how unsurgical is such a procedure. When one takes up a discussion of drainage of these gravely ill patients, each manipulation of the operator should be performed with the idea of drainage.

Each adhesion broken is drainage, each partial and complete bowel obstruction released is drainage, each distal abscess exposed is drainage, each structure released from its fixed and dependent point and elevated is drainage. The elevation of the peritonitic and half paralyzed bowel by the proper system of drainage improves its circulation and early encourages bowel movement.

Drainage must be directed toward the mucous membrane of the bowel as well as its peritonitic surface and therefore the necessity of releasing all partial bowel obstructions. In many cases it is necessary to open the bowel in one or more places in order to relieve distention, as the distended bowel must never be returned to the abdominal cavity.

Taking the position that the final and fatal dose of toxins most often comes from absorption of infectious fluids from the mucous membrane of the obstructed bowel, this source of infection must entertain our early consideration in the drainage of these cases. We know if the peritonitic patient develops a fistulous opening from the bowel, such drainage will often save the patient and a like fistula is often produced as an operative step in the worst cases.

It is a violation of all sound surgical principles for any operator to attempt this radical surgery of evisceration of the peritonitic structures and then attempt to drain by any kind of tubal drainage; the coffer-dam of gauze must be used.

The result of this radical method of treatment of the peritonitic patient will depend in a large measure on the ability of the operator to release the fixed structures from their anchored point.

Evisceration must be done with the greatest gentleness and never by making traction on the bowel, which is a source of profound shock. Just sufficient traction is made on the bowel to define its fixed point and then the structure is released by gently insinuating the finger beneath the fixed structures and in this way the bowel is, so to speak, lifted from its fixed point.

The proper method of inserting the through and through suture of silkworm gut should be followed; the terraced method of closing or partially closing the abdominal wall in the peritonitic patient should never be done. The through and through suture is a drainage one and is never followed by the explosion of the abdominal incision, nor do you ever see a serious continued infection of the abdominal wall which has a mortality the world over in these cases in which the terraced method of suture is used.

The coffer-dam system of drainage cannot be instituted unless the source of the peritonitis is removed and all adhesions broken. The surgeon must prepare a residence for the drain ere it can be inserted. The coffer-dam has its most worthy purpose, quite foreign to drainage per se; its

mechanical elevation of the peritonitic structures, preventing them from collapsing into dependent and infected areas and thus producing a postoperative bowel obstruction, is its most valuable function.

Further, it is useless to place the coffer-dam of gauze on top or in the neighborhood of remaining pathologic structures, nor can one eviscerate 5 feet of peritonitic bowel leaving the sixth foot fixed, which becomes the course of a postoperative bowel obstruction.

To begin a radical toilet commands the surgeon to travel to the limits of the peritonitic structures. I have seen but one postoperative bowel obstruction follow this radical surgery and drainage by the coffer-dam.

It is useless to compare this with the multiple postoperative complications which follow inferior primary work in the peritonitic abdomen. I am unable to find record of a postoperative pneumonia in the Joseph Price Hospital following this radical work in peritonitis.

I have illustrated elsewhere each step in the toilets of the peritonitic abdomen and insertion of the coffer-dam system of gauze, which must be done as a perfect piece of civil engineering.

By whom must this surgery be done? Certainly not by the beginner, but the difficulties in performing thorough work must not deter the teacher from placing this condition on a sounder surgical basis.

SUMMARY

The physiological surgeon's practice of adding more days of delay to the already too late patient is a most disastrous way of teaching the general profession earlier work.

Any attempt to teach the general profession that there is a time to operate and a time to wait, can never meet with good results in a condition whose signs and symptoms are not proportionate to the underlying pathological lesion.

The classification of the peritonitic patient into operative and non-operative hours is moving all acute perforative lesions to a later operative hour.

The quiescent stage of the physiological surgeon is not a subsidence of a diffuse or general peritonitis, but is the subsidence of a very local condition.

The physiological surgeon's determination as to the extent of the peritonitis following a ruptured appendix is based upon a false hypothesis, in that general tenderness and distention do not mean general peritonitis.

We have a great number of times operated on the physiological surgeon's patient who had been put on the waiting list on account of a supposed diffuse peritonitis and have proved to all present that the patient never had a peritonitis.

The peritonitis is not the only source of toxemia or infection which is to be considered following the perforation of any abdominal organ.

A peritonitis following a perforated appendix has its protective elements as well as its toxemic and the peritonitic endothelial membrane has its powers of absorption of toxins reduced to a minimum.

The peritonitis is physiological as well as pathological and were it not for the fact that the peritonitis superimposes other sources of toxemia upon itself, such as distal abscesses, bowel obstruction, etc., it would often win the fight.

The Fowler position does not establish the most dependent point for drainage, as we have attempted to illustrate in figures which accompany this article.

The Fowler position ignores the feeble and toxic heart of the peritonitic patient who should be placed in the position of flexion and rest.

There is not a free circulation of fluids in the peritonitic abdomen which is divided into many compartments by the plastic peritonitis which prevents gravity of such infected fluids to the pelvis of the patient in the Fowler position.

The lymphatics are not the true absorbents of the peritoneum, but the blood

vessels are and there is little, if any, difference in the fertility of the absorbents of the upper and lower abdominal regions, which is one of our reasons for not endorsing the Fowler position.

Gravity is of little importance in the peritonitic abdomen which is demonstrated by finding the pus in that particular location of the abdominal cavity where the appendix is located and not in the pelvis as one would expect from placing the patient in the Fowler position.

Tubal drainage is inferior drainage as compared with gauze and drainage of the peritonitic abdomen cannot be dismissed as drainage per se, but the mechanical function of the coffer-dam of gauze as a drain is most important and absolutely necessary.

The drainage must be conducted so as to drain the mucous membrane of the obstructed bowel, the absorption of toxins from which is often the cause of the final and fatal dose of infection; therefore the obstructed bowel must be released from its fixed point and often punctured in one or more places and in some cases producing a therapeutic fistula.

All kinds of drainage are by necessity local after the first few hours following the operation, but gauze has many more times the area of surface drain than that of any other kind of drainage.

Gauze drainage must be inserted as a continuous and solid wall, two or more columns of gauze are never inserted, as such flying buttresses of gauze predispose to postoperative bowel obstruction.

We never insert a drain to a clean zone of the abdominal cavity anticipating on account of its dependent point that it may later become pus laden.

Postoperative complications very rarely follow thorough primary work; postoperative bowel obstruction, distal abscesses, harmful adhesions, metastatic conditions, etc. are the penalties of incomplete primary work.

The peritonitic patient from a ruptured appendix rarely dies from the peritonitis per se, but bowel obstruction is probably the cause of the final and fatal dose of toxemia.

The surgical pathology of peritonitis will have to be rewritten in that the teaching thus far has been directed toward the peritonitis per se, ignoring all complications.

The peritonitic peritoneum little absorbs infected fluids.

The treatment of appendicitis of perforative origin is drainage.

The principle on which intra-abdominal drainage is established is the release of intra-abdominal tension. Excessive intra-abdominal tension injures the protective influence of the epithelium of the peritoneum and thus increases peritoneal absorption.

In fact, all the surgical steps in the treatment of the peritonitic abdomen are based on the broadest principles of drainage, such as breaking of all adhesions, exposure of abscessed cavities, release of partial and complete bowel obstruction plus the removal of all gangrenous structures including the removal of the primary source of infection which is the very foundation of drainage.

SOME REMARKS ON THE

INTERRELATION OF GYNECOLOGICAL AND GASTRO-INTESTINAL SYMPTOMS*

G. L. MOENCH, M.D.

NEW YORK CITY

THE trite saying that one should treat not the organ or the disease but the patient applies perhaps nowhere more succinctly than in the field of gynecology. Symptoms involving other organic systems are very frequently present in gynecological cases. I remind the reader here only of the various symptoms involving many different organs which are seen so frequently with menstruation and pregnancy. Thus the gynecologist should know at least sufficient general medicine to be able to interpret correctly symptoms and signs of nongenital origin, so as to be able to advise and treat his patients intelligently. To pass up the responsibility by immediately and uncritically referring all such patients to the respective specialists may easily result in a wrong perspective in which each man sees but a small part of the entire clinical picture, reminding one of the three blind men who examined the elephant. An example of such overspecialization is a case I saw at my office where the woman had been treated for several months for gastritis and indigestion because of nausea and vomiting, and the main fact that she was pregnant had been entirely overlooked.

As in pregnancy, so in many other gynecological lesions and disturbances, gastrointestinal symptoms are especially common, and I shall limit myself here to this interrelation of gynecological and gastrointestinal symptoms, and even here stress only some more or less common associations.

Considering the close proximity of the genital organs to all others found in the abdominal cavity and the interrelation of the blood supply, general, and autonomic nervous innervation, it would indeed be strange if disturbances in the genital system did not affect the gastrointestinal system, and vice versa. That the correct interpretation of the mechanism of the influence of one of the systems on the other is, however, not always easy is shown by the fact that the vomiting of pregnancy, for example, has been explained on a purely mechanical basis, due to the enlarging uterus, on the physical basis of changed blood supply, on a nervous basis, due to the stretching of the peritoneum, on a chemical basis, as the result of absorption of fetal toxins by the intestines or general system, and on a psychic basis, as a repugnance and revolt of the woman against carrying in her body a part of a consciously or subconsciously disliked male partner.

There are, of course, many lesions where the interrelation of genital and gastrointestinal symptoms is at once evident. That markedly relaxed abdominal walls and relaxation of the pelvic floor may cause gastrointestinal symptoms goes without saying, and I have cured many such cases by perineal repair and a proper belt. In the same way, large fibromyomata, ovarian and other pelvic tumors may cause gastrointestinal symptoms by causing changes in abdominal pressure. Appendicitis may lead to perisalpingitis and actual salpingitis and the other way round. At the same time every case of pain in the right lower quadrant, especially if the pain comes and goes, or lasts only minutes or an hour or so, and is not accompanied by fever, is not appendicitis, though often diagnosed as such and uselessly operated for. Spasms of the intestines, cramps, or dilatation of the cecum due to gas from intestinal fermentation or air swallowing must always

* Presented at a meeting of the N. Y. Medical Union.

be excluded. Again, adnexitis often leads to adhesions to the intestines, sigmoid and rectum, and so results in disturbances of intestinal function of various types, such as spasms and even intestinal obstruction. Even where no adhesions to the intestines or to the rectum are present, pelvic inflammation may cause spastic contractions of the intestines and tenesmus. Again, intestinal disturbances may lead to pelvic congestion. Constipation alone is often an important factor in pelvic congestion and some cases of pelvic pain can be cured readily by overcoming such constipation. The relation of mucous colitis to the genital system is also very close and may lead to a multitude of symptoms referred to the genital tract.

Personally I feel that mucous colitis is regarded too much as a clinical entity. Perhaps there is here an hereditary or congenital irritability and supersensitiveness, but I feel that genital inflammation can produce exactly the same symptoms, and following such inflammation there often remains an irritability of the colon which only long-continued diet can overcome. There are, however, undoubtedly also nervous factors involved. One of my patients who came to me with bilateral masses has been entirely relieved of her pelvic inflammation by conservative measures, but gets low abdominal distress and pains in the adnexal regions following indiscretions in diet, or as the result of emotional upsets. At times her menstrual cycle is also affected by these upsets and it is well known that disturbances of the abdominal blood supply may cause menstrual abnormalities as well as abortions and even premature labor.

Being a firm believer in the importance of psychic factors in the production of some peptic ulcers, I think that even in such cases at times a relationship between genital and gastrointestinal systems will be discoverable.

Thus in every female patient with gynecological or gastrointestinal symptoms or both, we must determine first if an organic lesion is present, and second, where it is situated. A careful general and pelvic examination, and, if necessary, gastric analyses, gastrointestinal *x*-ray pictures, laboratory examinations of blood, urine, etc., will usually determine this. At times a basal metabolism test will show a hyperthyroidism which may be responsible for the gastrointestinal disturbances, and an electrocardiogram may discover a heart lesion which has been hiding behind gastrointestinal symptoms. The gastric crises of locomotor ataxia must also be mentioned here. Sometimes a cystoscopy revealing a markedly trabeculated bladder may be the first sign-post leading to a correct diagnosis; once again showing the interrelation of various symptoms and signs in different organ systems. One must, however, always remember that finding a gynecological lesion does not preclude the presence of gastrointestinal disease and vice versa.

After an organic lesion has been found, we must carefully evaluate whether it can explain *all* the symptoms the patient complains of, and if the symptoms referred to one organ system can be explained by the organic change present in the other. Here it must not be forgotten that the gynecological lesion, for instance, may obscure or hide gastrointestinal symptoms or contrarily cause only gastrointestinal disturbances, or that the gastrointestinal lesion may produce only gynecological symptoms. Finally, if organic changes are found in both gynecological and gastrointestinal systems, it must be determined whether or not the two lesions are separate entities or whether they are interrelated and if the latter, in what way.

Despite experience and careful examinations, mistaken diagnoses are not uncommon. Perhaps a few illustrative cases from my own practice may be of interest here.

Case 1. A white female, thirty-six years old, complained of backache, a feeling of "losing insides," "indigestion" and gastrointestinal cramps. Examination revealed relaxed abdominal walls and vaginal prolapse. No gastrointestinal lesions were found. Operative repair

and a belt helped the patient, but her intestinal cramps and "indigestion" persisted. I then thought of the possibility of ureteral stricture and one was found on the right side. Dilatation of the ureter immediately relieved the patient of all gastrointestinal symptoms. After three months a recurrence of the gastrointestinal symptoms was again cured by ureteral dilatation. The rôle of a ureteral stricture in producing gastrointestinal symptoms is perhaps not so well known, but should always be thought of. After all, Dietl's crises, resulting in abdominal pain and vomiting, have practically the same etiology except that the sudden obstruction of the ureter causes more acute and correspondingly more severe symptoms and signs.

Case II. White female, forty years old. The diagnosis was fibromyomata uteri with right-sided ovarian cyst. Operation showed uterine fibromyomata and normal ovaries, but an enlarged and markedly dilated cecum. (Aside from a dilated cecum, a dilated sigmoid may also lead to misinterpretation on vaginal palpation.)

Case III. White woman, forty-five years old, with menorrhagia. The cervix was small, leading directly into a hard nodular mass which moved freely with the cervix. The adnexal regions were negative. Diagnosis: Fibromyoma uteri. Operation showed a carcinoma of the sigmoid attached firmly to the atrophic uterine fundus. A two-stage Mikulicz operation was done.

Case IV. A thirty year old white woman complaining of pains in the left lower quadrant. On palpation a nodular, doughy tumor was felt in this region. Diagnosis: dermoid cyst. Operation showed only enlarged appendices epiploicae closely packed together in the left adnexal region.

Case V. Same symptoms, palpatory findings and diagnosis as in Case IV. Operation showed slight adnexitis and adhesions to a thick omentum lying low down in the pelvis.

Case VI. The last case is one in which gynecological findings were apparently associated with severe gastrointestinal disease. The diagnosis was fibromyomata of the uterus. The patient refused operation and was later taken to another hospital with very severe abdominal pains, distention, fever and leucocytosis. A diagnosis of acute pancreatitis was made, but at operation nothing was found, except the uterine fibromyomata. The patient died the next day. An autopsy showed a valve-like occlusion of the right coronary artery by a calcified arteriosclerotic plaque.

So much for cases in which organic lesions are found. At times, however, no organic lesions are discoverable on the most careful examination, or the lesions actually present cannot possibly explain the patient's gastrointestinal or gynecological complaints. What is to be our method of procedure in such cases? Shall we without further ado send such patients to the gastroenterologist or call them neurotic and hysterical and throw up our hands and pass them on to the neurologist? I think not. After all, there must be a cause for such symptoms and the gynecologist, first because he has already been in contact with the patient and secondly because of the peculiarly intimate nature of the relations between him and the patient, seems to me to be eminently fitted to obtain such accurate and detailed confidential knowledge as may assist in clearing up the condition.

It is here that a painstaking and tactful anamnesis is of inestimable value.

The onset and duration of the complaint, the amount of pain, its location, etc., the continued or periodical appearance of the symptoms, are all important. But further than this we must know of the patient's habits, her diet, bowel movements, her occupation, domestic and sexual life and mental state to be able to treat intelligently.

So-called indigestion with flatulence, belching, gastric distress or even marked pain are too often labeled astritis, hyperacidity or hypoacidity, with or without gastric analysis, and treated with all sorts of medication when the root of the whole trouble lies in domestic difficulties. Nostalgia or grief may cause gastrointestinal upsets as well as gynecological symptoms. Excitement of any kind may lead to constipation or diarrhea, and air-swallowing may be the sole cause for the eructations which are sometimes almost continuous. Even if determination of the gastric acidity reveals values above or below normal, this does not necessarily

mean that organic disease is present. Emotional disturbances alone suffice to produce such conditions. I have a patient who from time to time has all the signs of peptic ulcer, but only when domestic or other difficulties occur. Otherwise she has no complaints of any kind and can eat and drink whatever she pleases with no unpleasant effects. Such psychic effects are not at all uncommon. The secretions and excretions of the body: saliva, gastric juice, sweat, urine, feces, etc., are all to a much larger degree than is often realized under the control of the psyche. Everyone will remember numerous examples which prove this. Again nausea and vomiting may appear only around the menstrual period, perhaps due to pelvic congestion. In other cases the reaction is a psychic revolt against menstruation because of a desire for offspring. After coitus also sometimes gastrointestinal symptoms may come on. Disgust or even religious principles may be the underlying factors in such cases. In contradistinction to such emotional causes irregular hours, overeating, or the opposite, wrong diet, peculiar allergic sensitivity to certain foods, such as shell fish, various berries, pineapple, etc., alcoholic excess or overindulgence in tobacco, may be responsible. A change to a sedentary occupation may be the sole factor underlying constipation. Roentgen-ray treatment for uterine fibromyomata may cause diarrhea, as in a patient of mine who was treated by her family physician for this complaint, since the patient had not told him about her *x*-ray treatments, not connecting these in any way with her gastrointestinal symptoms. Asthenic conditions and even gonadal insufficiency coupled as it always is with other hormonal defects may cause symptoms simulating gastrointestinal disease. At the same time, we must not forget that chronic gall-bladder disease or appendicitis, even if mild, may be the root of the trouble. Intestinal parasites also must be thought of, though I believe the presence of a small number of parasites does not immediately explain the whole problem. Often such parasites are purely coincidental.

With such a galaxy of causes for gastrointestinal distress in gynecological disease, it is evident that routine treatment is impossible. It is useless to medicate for gastrointestinal distress which is based on domestic, financial or other difficulties which the physician can only alleviate if he will take the time really to get at the source of the patient's troubles. Sometimes a mild sedative such as bromural will alleviate gynecological and gastrointestinal symptoms quicker than any complicated specialistic treatment. Again a sympathetic attitude and reassurance of the patient in regard to her gynecological troubles will with one stroke cure all gastrointestinal distress.

In some cases diet, of course, is important, but I feel we must be most individualistic here too. Diets and dieting to my mind play far too great a rôle in the daily life of the average American citizen. If marked constipation is present, regulation of diet, roughage, etc., is important, but with such roughage again we must avoid gastrointestinal irritation. Sometimes cathartics are necessary. Often they work well when combined, especially in the obese, with a cholegogue. Even in constipation, however, it must be remembered that patients differ, some naturally move their bowels twice a day, some once a day or once in two days, or even less often, without therefore really suffering from constipation. Rash interference in such cases can only lead to harm.

SUMMARY

I have reviewed here briefly some of the organic and psychic causes of the interrelationship of gynecological and gastrointestinal lesions and stressed the need of accurate diagnosis as a prerequisite for intelligent treatment. No attempt has been made to cover the entire field. My more or less random remarks simply record some of my personal experiences and my opinions based thereon. I hope that a full discussion will enable me to pick up many valuable points.

FULL TERM INTRA-ABDOMINAL PREGNANCY

ALFRED M. HELLMAN, M.D., F.A.C.S. AND HERBERT J. SIMON, M.D.

Attending Gynecologist and Obstetrician, Sydenham Hospital; Associate Gynecologist and Obstetrician, Lenox Hill Hospital

Assistant Adjunct Gynecologist and Obstetrician, Sydenham Hospital

NEW YORK CITY

EXTRA-UTERINE pregnancy that continues until the fetus is viable and terminates in the delivery of a living child, is sufficiently rare to warrant the report of the case detailed below. A survey of the literature revealed that no complete bibliography on the subject had been compiled since Sittner* wrote his last paper in 1906. It was decided to try to bring Sittner's work up to date.†

The treatment of the placenta is discussed by every author on this subject. After a perusal of hundreds of reports and a consideration of the results obtained, one comes to the conclusion that the management of the placenta should depend on the findings in the individual case. Should the placenta be easily removable, as in the senior author's case, then complete excision is mandatory. On the other hand, should it be intimately attached to the intestines and other abdominal viscera, marsupialization or partial closure with drainage is the method of choice. Often the abdomen is closed completely, leaving the placenta in situ to absorb. There is no single rule for the management of the placenta, but the procedure in each case must be left to that indefinable something referred to as "surgical judgment."

Another point that one finds much discussed in the literature is, when to operate once the diagnosis is definitely established. Some say at once; others would delay until the fetus is viable; and still a third group advocate waiting for the death of the fetus so that placental separation may have begun. The only group with whom we take positive issue is the last. To wait for fetal death and beginning placental separation means to wait for maceration, and this unquestionably adds to the danger of sepsis and, of course, sacrifices the fetus. Again it is our opinion that each case must be treated according to its most pressing indications.

It is often said that the fetus need not be considered in extra-uterine pregnancy since it is so frequently deformed. While it is true that the chances of minor or major deformities are materially increased by gestation outside the uterus, nevertheless it is our impression that one finds perfectly normal infants in extra-uterine pregnancy much more often than some authors would have us believe. We regret that no exact percentage can be given, because in many of the cases reviewed the authors were either indefinite or failed entirely to mention the status of the newborn.

We have collected from the literature 311 cases of intra-abdominal pregnancy and are adding 5 cases now for the first time reported. The history of the senior author's case is given in full, the existence of the other four is noted but they are not described. This makes a total of 316 cases in which a living infant was delivered. This does not mean that the child was viable in each case. It would perhaps be better to include only truly viable fetuses, but Sittner, who has been considered the authority on this subject, has included in his series fetuses of twenty-two weeks' gestation or over. We have, therefore, included all of Sittner's cases, but since 1906 we have put into this series only cases in which the fetus was of at least twenty-eight weeks' gestation.

* *Arch. f. Gynäk.*, 64: 526–669, 1901. *Zentralbl. f. Gynäk.*, 27: 33–48, 1903. *Deutsche Med. Wchnschr.*, 29: 743; 759; 787, 1903; 32: 1200–3, 1906. *Die Heilkunde*, 12: 201–209, 1908.

† A conscientious effort has been made to include every case in the literature; however, any case omitted and drawn to our attention will be greatly appreciated. (An occasional reference to a case was found which could not be investigated because the journal was not in the Library of the N. Y. Academy of Medicine.)

Of the 316 cases we have studied, in 158 the fetus lived. By "lived" we mean survived eight days or longer. It was necessary to adopt some arbitrary standard to indicate true viability of the fetus and eight days was considered sufficient. Of the 316 mothers 212 lived, 101 died, and the outcome in 3 is unknown. We believe the principal causes of death for the mother are (1) peritonitis, (2) hemorrhage with shock, and (3) secondary factors including diseases of kidneys and heart, and later complications such as pneumonia.

In the whole series there are only 80 cases in which both mother and child survived, and of these 80 it is probable that the mother and child were not entirely well in more than 50. This figure is not arrived at by any true statistical study but rather by the impression one gets from the review of hundreds of these cases. It is impossible to say in how many cases the outcome was as fortunate as in the senior author's for in many full information is not given.

Of the 316 cases the infant was extracted per vaginam in two and the rest were removed by laparotomy. We have found 2 cases of combined intra-abdominal and intra-uterine pregnancy. In both cases the mother as well as the intra-uterine and intra-abdominal infants survived.

The following is the history of the senior author's case:

The patient was a para 1, gravida 1, twenty-five years of age, married one and one-half years. Her menses started at thirteen and were irregular up to two years before pregnancy, when the condition partially corrected itself. The past history included only rheumatism in childhood and an appendectomy at the age of fifteen. The first day of her last period was December 6, 1932, so that the computed date of confinement was September 13, 1933. She was first seen in the office of Dr. H. on September 11, 1933. She stated at that time that she had had an essentially normal pregnancy. Her blood pressure was 118/80. She had a marked presystolic blow at the apex, some dyspnea and slight edema of the ankles. Her urine was negative. Pelvimetry was normal. At the clinic where she had had her prenatal care she was told she might need a cesarean section for malposition of the fetus, and, thereupon decided to seek further advice and consulted the senior author. The cervix was soft and long, the uterus enlarged to the size of a twelve weeks' pregnancy, and floating above it in the transverse position was a good-sized fetus with the head in the right flank and the buttocks to the left. The patient was told that indeed a cesarean section was inevitable, but that labor should not be waited. A laparotomy was performed the following morning, September 12, 1933. The amniotic sac was directly beneath the abdominal wall. After freeing a few adhesions between the intestines and the sac, the latter was incised, whereupon there was an escape of a large quantity of rather dark amniotic fluid. The fetus was extracted without difficulty. The placenta was found attached to the posterior surface of the uterus and the posterior surfaces of both broad ligaments. With only slight bleeding the placenta was easily freed from the uterus and the right broad ligament. However, it was firmly adherent to the left tube and ovary and the left broad ligament. Therefore a left salpingo-oophorectomy was done and the placenta was thus removed complete with a minimum of bleeding. The wound was closed in layers without drainage. The infant was a female weighing 8 pounds 8 ounces, perfectly normal in all respects. Mother and child were discharged from the hospital on the tenth postoperative day, both in excellent condition and were both known to be doing well April 23, 1935.

SUMMARY

The important points in the management of extra-uterine pregnancy with a viable fetus have been discussed. Three hundred eleven cases have been collected from the literature and 5 new cases added, the senior author's case being the only one fully described. The additional 4 cases not described in detail have never before appeared in the literature. The information regarding them has been given to us by the doctors who had them in their charge. Permission to cite them has been given by: Drs. A. C. Posner and Carl Eggers, New York; and Drs. George Nordlinger and Raymond T. Holden, Jr., Washington, D. C.

A complete table including in all 316 cases is appended.

Summary of Cases

Date of Operation	Operator	Place of Operation	Result to Mother	Status of Fetus	Special Notes and Bibliography
Nov. 1809	Müller	Halbeau, Germany	Recovered	9 mo. fetus	Thèse de Paris, 1894
Aug. 19, 1813	Brükert	Berlin	Died	9 mo. fetus, lived	*Mag. f. d. ges. Heilkunde-Rust*, 3: 1, 1819
Dec. 8, 1814	Domenico Novara	Porto Maurizio	Died	9 mo. fetus, lived	*J. Univ. d Sc. Med.*, 3: 119, 1816
1816	John King	So. Carolina	Recovered. Operation per vagina	Full term Lived	*New York Med. Rep.*, p. 388: 1817
1827	Mattfeld	Tubingen	Died (placenta left in situ)	Lived	*Neu Ztschr. f. Geburtsh.*, 1: 134, 1834
Sept. 15, 1837	Zwanck	Hamburg	Recovered	Probably full term, lived	Abst. *Lancet*, 2: 801, 1837–8
Mar. 1, 1841	Hauff	Germany	Died	35 wk. fetus, died in 50 hrs.	*Med. Ann. Heidelberg*, 8: 439, 1842
1852	Prof. Pietro Lazzati	Milan	Died	9 mo. fetus, alive but did not respire	Manuele del Porto Mechanico ed° Instrumentale del Lovati, Milano, p. 194, 1854.
Mar. 27, 1863	Prof. Eugène Koeberlé	Strasbourg	Died	9 mo. fetus, died second morning	*Gaz. Med. de Strasbourg*, 10: 160, 1863
Apr. 21, 1864	Dr. Robt. Greenhalgh	London	Died	9 mo. fetus, died in a few minutes	*Med. Mirror*, p. 689 (Nov.) 1864
Mar. 3, 1870	Sale	Aberdeen, Miss.	Died	Lived 6 mo.	*New Orleans M. & S. J.*, p. 727: 1870
Dec. 1871	L. Bandl	Vienna	Dead at operation	Lived 10 mins.	*Wien. med. Wchnschr.*, 8: 8, 1874
Oct. 5, 1872	John Scott	London	Died	30 wk. fetus, lived two days	*Tr. Obst. Soc. London*, 15: 309, 1873
Aug. 14, 1875	Th. P. Jessop	Leeds, England	Recovered	33 wk. fetus, lived 11 mo.	*Tr. Obst. Soc. London*, 18: 261, 1876
Nov. 5, 1876	Prof. Otto Spiegelberg	Berlin	Died	40 wk. fetus, lived 3 mo.	*Arch. f. Gynäk.*, 13: 74, 1879
May 25, 1877	Heywood Smith	London	Died	9 mo. fetus, lived 30 min.	*Tr. Obst. Soc. London*, 20: 5, 1878
Nov. 5, 1877	Henry Gervis	London	Died	36½ wk. fetus, lived 6 hrs.	*Brit M. J.*, 2: 884, 1877
Aug. 19, 1878	Ernst Frankel	Breslau	Died	33½ wk. fetus, lived 24 hrs.	*Arch. f. Gynäk.*, 14: 197, 1879
May 29, 1879	Carl Schroeder	Berlin	Died	34 wk. fetus, lived	*Ztschr. f. Geburtsh. u. Gynäk.*, 5: 115, 1880
June 29, 1879	M. Hofmeier	Berlin	Died	Lived	*Ztschr. f. Geburtsh. u. Gynäk.*, 5: 115, 1880
Nov. 1879	M. A. Pallen	New York	Died	26 wk. fetus, lived a few min.	*Am. J. Obst.* (July) 1880
Dec. 19, 1879	B. Christian Vedeler	Christiania	Died	35 wk. fetus, died next day	*Norsk Mag. f. Laegevidenskaben* 6: 86 (Juni) 1880
Jan. 10, 1880	C. C. Th. Litzmann	Kiel	Died	39½ wk. fetus, lived 15 min.	*Arch. f. Gynäk.*, 16: 362, 1880
Jan. 31, 1880	Lawson Tait	Birmingham	Died	9 mo. fetus, lived	*Obst. J. G. B. & Ireland*, 2: (Oct.) 1880
May 11, 1880	Henry P. C. Wilson	Baltimore	Died	Full term fetus, lived 18 mo.	*Tr. Am. Gynec. Soc.*, 6: 461, 1882
July 26, 1880	W. Netzel	Stockholm	Died	Full term fetus, lived 48 hrs.	*Hygiea* (Stockholm) 18: 169, 1881
May 1881	W. Goodell	Philadelphia	Died	28 wk. fetus, lived a few min.	*Am. J. Obst.* (Oct.) 1881
June 28, 1881	Matheison	Ontario, Eng.	Recovered (Vaginal operation)	Full term fetus, lived	*Brit. M. J.*, 1: 999 (May 24) 1884
July 9, 1881	August Martin	Berlin	Recovered	7 mo. fetus, did not breathe but cord pulsated	*Berl. Klin. Wchnschr*, 18: 753 (Dec. 16) 1881
July 13, 1881	Guiseppe Beisone	Pinerolo, Italy	Died	9 mo. fetus, lived	*Gaz. Med. di Torino*, 32: 553–557, 1881
Feb. 15, 1882	Hildebrandt	Konigsberg	Died	Full term fetus, lived	*Berl. klin. Wchnschr.*, 29: 465 (July 20) 1885
Oct. 3, 1882	Hildebrandt	Konigsberg	Died	8 mo. fetus (asphyxiated & not resuscitated)	*Berl. klin. Wchnschr.*, 29: 465 (July 20) 1885
Jan. 1884	R. Frommel	Erlangen	Died	24 wk. fetus, lived few min.	*München. med. Wchnschr.*, p. 4, 1892
"About" 1884	C. S. Bonifield	Cincinnati	Died	6 to 7 mo. fetus, lived or died?	*Cincinnati Lancet Clinic*, p. 66 (July 16), 1910
June 6, 1885	J. Williams	London	Recovered	8¾ mo. fetus, lived few min.	*Trans. Obst. London*, 29: 482, 1888
Nov. 4, 1885	Lazarewicz	Kharkof	Recovered	7 mo. fetus, lived 21 days	*Urach*, St. Petersburg, 7: 76–115, 1886. Also in: *Rep. Univ. de Nouv. Arch. d'Obst. et de Gynec.*, pp. 277–279 (July 25) 1886

SUMMARY OF CASES (*Continued*)

Date of Operation	Operator	Place of Operation	Result to Mother	Status of Fetus	Special Notes and Bibliography
July 29, 1886	A. Stadtfeldt	Copenhagen	Died	Full term fetus, lived	*Tidende*, p. 889 (Sept. 22) 1886
Oct. 19, 1886	H. Champneys	London	Died	7 mo. fetus, lived few min.	*Trans. Obst. London*, 29: 456, 1888
Mar. 30, 1887	Jos. Price	Camden, N. J.	Died	7½ mo. fetus, lived, 4 hrs., sl. deformed	Communicated by the operator to Sittner Oct. 21, 1900. (Unpublished)
May 29, 1887	H. Treub	Leyden	Recovered	8¼ mo. fetus, lived	*Ztscb. f. Geburtsb. u. Gynäk.*, 15: 384, 1888
June 26, 1887	Prochownich	Hamburg	Died	8 mo. fetus, lived 24 hrs.	F. Spaeth: *Ztscbr. f. Geburtsb. u. Gynäk.*, 16: 269, 1889
Sept. 1887	L. Brühl	Bern	Recovered	22 wk. fetus, lived a min.	*Arcb. f. Gynäk.*, 31: 404, 1887
Oct. 29, 1887	Breisky	Vienna	Recovered	8 mo. fetus, lived 19 days	*Wien. med. Wcbnscbr.*, p. 48, 1887
Mar. 22, 1888	Lebec	Paris	Died	8 mo. fetus, lived 24 hrs.	*Bull. et mém. Soc. d. cbir. de Par.*, 17: 274, 1891
July 10, 1888	Eastman	Indianapolis	Recovered	Lived, 8 mo.	*Am. J. Obst.* (Sept.) 1888
Oct. 12, 1888	von Braun Fernwald	Vienna	Died	8½ mo. fetus, lived	*Arcb. f. Gynäk.*, 37: 286, 1890
Nov. 1, 1888	Olshausen	Berlin	Recovered	9 mo. fetus, well at 1 yr.	*Deutscbe med. Wcbnscbr.* vol. 9: 1890
Feb. 11, 1889	von Braun Fernwald	Vienna	Recovered	9 mo. fetus, lived 12 hrs.	*Arcb. f. Gynäk.*, 37: 286, 1890
Feb. 27, 1889	Olshausen	Berlin	Recovered	8½ mo. fetus, lived 1½ hrs.	*Deutscbe med. Wcbnscbr.*, vol. 9: 1890
Sept. 22, 1889	Negri	Venice	Recovered	8 mo. fetus, lived 18 hrs.	*Rev. Ven. di Sc. Med.*, p. 10, (Gennaio-Febbra) 1890
Oct. 9, 1889	Pearce and Gould	London	Died	12 mo. fetus? Signs of life	Kelly: Operative Gyn., 1898, 2: 458
Oct. 18, 1889	M. Treymann	Riga	Died	24 wk. fetus?	*St. Petersburger med. Wcbnscbr.*, p. 141, 1890
1889	J. H. Carstens	Detroit	Died	6 mo. fetus, lived 1 hr.	Communicated by the operator to Sittner. (Unpublished)
1889	E. Tauffer	Budapest	Recovered	4 to 5 mo. fetus, said to have breathed	Toth: *Arcb. f. Gynäk.*, 51: 410, 1896
Jan. 1890	P. Zweifel	Leipsig	Died	Lived a few minutes	*Arcb. f. Gynäk.*, 41: 1, 1891
Feb. 4, 1890	Geo. Rein	Kiew	Recovered	9¼ mo. fetus, lived	*Zentralbl. f. Gynäk.*, p. 50: 1892
Apr. 25, 1890	Galabin	London	Died	7 to 8 mo. fetus, lived 6 wks.	(After Harris) *Monatscbr. f. Geburtsb. u. Gynäk.* (Aug.) 1897
June 16, 1890	Chrobak	Vienna	Recovered	7½ mo. fetus, lived 24 hrs.	*Wien. klin. Wcbnscbr.*, p. 10, 1891
June 24, 1890	J. Taylor	Birmingham	Recovered	Full term fetus, sl. deformed, lived 5 mo.	*Trans. Obst. Soc. London*, 33: 115, 1891
Aug. 16, 1890	Negri	Venice	Died	8 mo. fetus, lived 2 days	Communicated by operator to Sittner. (Unpublished)
Aug. 21, 1890	Schoonen	Antwerp	Recovered	8 mo. fetus, lived few min.	*Bull. Soc. Belge de Gynec. et d'obst.* 2: 142–149, 1891
Nov. 1890	Rosenstein	Breslau	Died	35 wk. fetus, lived 8 days	*Monatscbr. f. Geburtsb. u. Gynäk.*, 19: 124, 1904
Dec. 1890	Geo. Rein	Kiew	Died	21 wk. fetus, lived a few min.	*Zentralbl. f. Gynäk.*, 16: 969, 1892
Jan. 1891	Fr. Schauta	Vienna	Recovered	21 wk. fetus, took a few breaths	Beiträge zu Casuistik, Prognose u. Therapie der Extrauterine Schwangerschaft, Prague, 1891
Feb. 2, 1891	W. Körte	Berlin	Recovered	7½ mo. fetus, lived 3 hrs.	Schneider: *Deutscbe med. Wcbnscbr.*, 37: 882, 1892
Feb. 12, 1891	Gueniot	Paris	Died	7½ mo. fetus, lived 15 min.	Reported in the Soc. Obst. de France, 1891
June 6, 1891	Frommel	Erlangen	Recovered	9 mo. fetus, lived 4 mo.	*München. med. Wcbnscbr.*, p. 1, 1892
June 16, 1891	Stevenson	Cape Colony	Recovered	8½ mo. fetus, lived 48 hrs.	*Trans. Obst. London*, 35: 175, 1893
June 1891	Marchand	Paris	Recovered	24 wk. fetus, lived 1½ hrs.	*Gaz. des bóp.*, 65: 546–48 (May 17) 1892
Aug. 1, 1891	G. Novy'	Prague	Recovered	33 wk. fetus, lived 24 hrs.	*Casop. lék. česk.*, 971, 1905
Aug. 1891	Schoonen and Rochet	Antwerp	Recovered	Lived 3 days	*Bull. Soc. Belge de Gynec. et d'obst.*, p. 5, 1891
Oct. 1891	H. Fritsch	Bonn	Recovered	29 wk. fetus, died at operation	Gyn. operations, 1891–92
Nov. 19, 1891	von Strauch	Moscow	Recovered	9 mo. fetus, breathed and died	*St. Petersburger Med. Wcbnscbr.* (Aug. 15) 1892
Mar. 1892	R. Kaltenbach	Halle a.S.	Recovered	32 wk. fetus, lived 1 hr.	v. Herff: *Ztscbr. f. Geburtsb. u. Gynäk.*, 34: 12, 1896
Apr. 27, 1892	Handfield-Jones	London	Died	8 mo. fetus, lived 6 mo.	*Lancet*, 2: 972, 1895
Apr. 29, 1892	Sippil	Frankfort a.m.	Died	7 mo. fetus, lived 5 days	*Deutscbe med. Wcbnscbr*, p. 37, 1892

Summary of Cases (*Continued*)

Date of Operation	Operator	Place of Operation	Result to Mother	Status of Fetus	Special Notes and Bibliography
Oct. 23, 1892	Price	Philadelphia	Recovered	40 wk. fetus, well at 5 yrs.	*Trans. State Med. Soc. Penn.*, p. 152, 1893
Dec. 29, 1892	Marchand	Paris	Died	9 mo. fetus, lived 8 mo.	Orrilard, Thèse de Paris, 1894
Mar. 1893	E. Tauffer	Budapest	Recovered	27 wk. fetus, lived only a short time	Toth: *Arch. f. Gynäk.*, 51: 410, 1896
Apr. 15, 1893	Sneguireff	Moscow	Died	9 mo. fetus, lived 18 mo.	Kelly: Operative Gyn., 1898, 2: 458
Apr. 1893	W. T. Lusk	New York	Recovered	7 mo. fetus, lived 26 min.	*N. Y. J. Obst. & Gynec.* (July) 1893
May 12, 1893	Urbain	LaBouverie	Recovered	9 mo. fetus, lives "today"	*Bull. de la Soc. Belge de Gyn. et d'Obst.*, 1893
Aug. 25, 1893	Mc.Nutt	Oakland, Calif.	Died	9 mo. fetus, lived a few min.	*J. A. M. A.*, 23: 278 (Aug. 18) 1894
Aug. 29, 1893	E. Regnier	Vienna	Recovered	9 mo. fetus, born alive but did not breathe	*Zentralbl. f. Gynäk.*, p. 1170, 1893
Sept. 11, 1893	Roncaglia	Modena	Recovered	7½ mo. fetus, lived 23 days	*Ann. di ostet. e. ginec.* (May) 1895
Oct. 15, 1893	Treub	Leyden	Recovered	8½ mo. fetus, lived 50 hrs.	*Bull. et mém. Soc. Obst. et Gyn. de Paris*, p. 40, 1894
Dec. 2, 1893	Hofmeier	Würzburg	Recovered	9 mo. fetus, born alive but did not breathe	Anatomische u. Klinische Beiträge zu Lehre von der Ectopischen Schwangerschaft, Würzburg, 1894
Dec. 18, 1893	Gueniot	Paris	Recovered	9 mo. fetus, lived 16 days	Reported in the Academie de Med., 1894; also a letter to Sittner dated Oct. 3, 1900.
1893	P. Horrocks	London	Died	Lived only a few mins.	*Trans. Obst. London*, 35: 180, 1893
Jan. 13, 1894	Cullingworth	London	Died	Lived, 7½ mo.	*Brit. M. J.*, 22: 12, 1894
Feb. 1894	B. Braun	Vienna	Died	28 wk. fetus, died quickly	*Zentralbl. f. Gynäk.*, p. 485, 1894
Feb. 1894	S. C. Gordon	Portland, Me.	Recovered	22 wk. fetus, took a few breaths	*Am. Gyn. & Obst. J.* 9: 28 (July) 1896
Feb. 7, 1894	H. Spencer	London	Died	27 wk. fetus, lived few min.	Letter from the operator to Sittner dated June 3, 1906
Apr. 4, 1894	Werder	Pittsburg	Recovered	8½ mo. fetus, lived 4 days	*Med. Record*, 24: 11, 1894
July 1894	McCosh	New York	Recovered	26 wk. fetus, lived 1 hr.	*Med. & Surg. Record, Presb. Hosp., N. Y.* (Jan.) 1896
Sept. 4, 1894	Potherat	Paris	Recovered	? wk. fetus, lived 4 wks.	*Bull. et mém. Soc. d. chir. de Paris*, 1896
Sept. 14, 1894	Tournay	.....	Recovered	9 mo. fetus, lived 2 hrs.	*Bull. de la Soc. Belge de Gyn. et d'Obst.*, 1894
Nov. 21, 1894	Eakins	Queensland	Died	8½ mo. fetus, lived	*Australian Med. Gaz.*, 15: 3, 1895
Dec. 10, 1894	Geo. Rein	Kiew	Recovered	30 wk. fetus, alive at 1 mo.	Harris: *Monatschr. f. Geburtsh. u. Gynäk.* (Aug.) 1897
?	A. Winter	Konigsberg i/Pr.	Recovered	21 wk. fetus, presume it could not live	By a letter to Sittner dated May 9, 1902 from Winter.
Feb. 3, 1895	Pestalozza	Florence	Recovered	9 mo. fetus, lived	*Ann. di Ost. e Gin.* (July) 1895
Apr. 1895	O. v. Herff	Halle a.S.	Recovered	33 wk. fetus, lived 3½ mo.	*Ztschr. f. Geburtsh. u. Gynäk.*, 34: 12, 1896
Apr. 1895	Snegnireff	Moscow	Died	38 wk. fetus, lived 1½ yrs.	Die Gebärmutterbluntungen, II Aufl., 1895
May 2, 1895	Pinard	Paris	Recovered	7 mo. fetus, alive at 2 mo.	*Ann. de Gynec.* (Aug.) 1895
May 1895	H. Cripps	London	Died	26 wk. fetus, lived only few min.	*Brit. M. J.*, 28: 3, 1896
Aug. 1895	R. Klein	Dresden	Died	39 wk. fetus, lived 15 mo.	By a letter to Sittner from the operator.
Sept. 20, 1895	Tauffer	Budapest	Recovered	9 mo. fetus, lived 4 mo.	Reismann: *Zentralbl. f. Gynäk.*, 20: 10, 1896
Sept. 1895	L. H Dunning	Indianapolis	Died	Fetus died during operation	Reported to the Marion County Med. Soc., Nov. 1895. Also by a letter from the operator to Sittner.
Nov. 18, 1895	A Gusserow	Berlin	Died	38 wk. fetus, lived	E. Runge: *Arch. f. Gynäk.*, 70: 690, 1903
Jan. 3, 1896	H. Spencer	London	Died	30 wk. fetus, lived 16 hrs.	By a letter from the operator to Sittner dated June 3, 1906
Jan. 1896	W. R. Wilson	Philadelphia	Died	Full term fetus, died during operation	*Am. Gyn. Obs. J.* 8: 605 (May) 1896
Feb. 20, 1896	N. Charles	Liege	Recovered	38 wk. fetus, "lives today"	*J. d'Accouch. de Belg.*, No. 49/51, 1897

SUMMARY OF CASES (*Continued*)

Date of Operation	Operator	Place of Operation	Result to Mother	Status of Fetus	Special Notes and Bibliography
Feb. 26, 1896	A. Martin	Rouen	Died	40 wk. fetus, lived 120 min., normal	*La Normandie med.*, 15: 4, 1896
Feb. 26, 1896	Hardie	Brisbane, Australia	Recovered	36 wk. fetus, lived 6 hrs.	*Brit. Gynec. J.* (Aug.) 1896
Feb. 26, 1896	Chrobak & Ludwig	Vienna	Recovered	9 mo. fetus, living at 4 wks. There was also a normally delivered intrauterine twin.	*Wien. klin. Wchnschr.*, p. 27, 1896
Mar. 1896	W Wilke	Hannover	Recovered	30 wk. fetus, lived 4 hrs.	*Zentralbl. f. Gynäk.*, 22: 1132, 1898
May 9, 1896	O. Semb	Christiana	Recovered	21 wk. fetus, lived ½ hr.	*Norsk. mag. f. Laegerindensk.*, 4R, Bd. 11, No. 9
Sept. 5, 1896	Karg	Zwickau	Recovered	32 wk. fetus, lived 2 days	Spörlin: *Zentralbl. f. Gynäk.*, 42: 1265, 1897
Sept. 1896	P. Sacchi	Cremona	Recovered	33 wk. fetus, lived 3 yrs.	*Gaz. Med. Cremon.* (May–June) 1897
Sept. 1896	A. Bertazzoli	Milan	Recovered	36 wk. fetus, lived few hrs.	*La Pratica Ostetr.-Ginecol.*, 1: No. 9, 16
Nov. 7, 1896	Ayers	New York	Died	7 mo. fetus, lived 3 wks.	Ayers: *Obstetrics* (Feb.) 1899; also *Med. News*, 17: 4, 1897
Jan. 1897	Jeannel & Crouzat	Toulouse	Died	34 wk. fetus, lived less than 1 day	*Ann. de la Soc. Obst. de France*, 1 Ser. 5 Vol. 1
June 11, 1897	Forster	Kansas City, Mo.	Died	9 mo. slightly deformed fetus, lived	*Homeopathic J. of Surg. & Gyn.*, 1: 49 (Jan.) 1898
Sept. 1897	Earnest	Atlanta	Recovered	32 wk. fetus, lived 1 hr.	*Ann. Gyn. & Ped.* (Jan.) 1898; also *Am. J. Obst.* (Jan.) 1898
Sept. 1897	Neugebauer	Warsaw	Recovered	37 wk. fetus, lived 9 hrs.	*Zentralbl. f. Gynäk.*, 22: 7851, 1898
Oct. 23, 1897	A. Bertazzoli	Milan	Died	22 wk. fetus, lived ½ hr.	*La Pratica Ostetr.-Ginecol.*, Anno 1, No. 9, 16
Oct. 1897	V. Kezmarszki	Budapest	Recovered	28 wk. fetus, lived 7 hrs.	By a letter to Sittner from the operator dated Oct. 29, 1900
Dec. 1897	C. v. Rokitansky	Graz	Recovered	36 wk. badly deformed fetus, lived	Jurinka: *Zentralbl. f. Gynäk.*, 22: 333 (Apr. 2), 1898
Jan. 1898	Campbell	Burban, S. Africa	Died	9 mo. fetus, lived ½ hr.	*Brit. M. J.*, 12: 3, 1898
June 1898	A. Bianchi	Bologna	Recovered	28 wk. fetus, lived 2 days	*Gaz. Med. Lombarda*, No. 41, 1899
July 1898	J. Bland Sutton	London	Recovered	50 wk. fetus, lived 2 hrs.	*Trans. Obst. London*, 40: 308, 1899
July 1898	P. Zweifel	Leipzig	Recovered	23 wk. fetus, lived 1 hr.	*Zentralbl. f. Gynäk.*, p. 1265, 1898
July 1898	A. Martin	Greifswald	Recovered	34 wk. fetus, lived 20 hrs.	*Monatschr. f. Geburtsh. u. Gynäk.* (June) 1899
July 1898	Kupferberg	Mainz	Recovered	36 wk. fetus, lived 3 wks.	Moebius: *Monatschr. f. Geburtsh. u. Gynäk.*, 15: 642, 1902
Oct. 1898	H. C. Dalton	St. Louis	Recovered	40 wk. fetus, lived 4 hrs.	*Am. J. Surg. & Gyn.* (Nov.) 1898
Oct. 1898	Boissard	Paris	Recovered	23 wk. fetus, lived only a short time	*Bull. de la Soc. d'Obst. de Paris*, 2: 14, 1899
Dec. 1898	Werder	.....	Died	36 wk. fetus, lived few hrs.	*Am. J. Obst.*, p. 796 (Nov.) 1908
	A. Winter	Königsberg i/Pr.	Recovered	25 wk. fetus, presume it did not survive	By a letter to Sittner
Feb. 1899	A. Muratoff	Kiew	Died	36 wk. fetus, lived	*J. akusb. i. zbensk. bolez.*, 13: No. 5, 1899
Feb. 1899	A. Benckiser	Karlsrube	Recovered	9 mo. fetus, lives "today"	By a letter from the operator to Sittner, dated May 2, 1906
Apr. 30, 1899	R. Chrobak	Vienna	Recovered	22 wk. fetus, presume it could not survive	L. Von Dittel: Beiträge z. Geb. u. Gyn. Festschrift für Chrobak, 1903
July 1899	N. Alexenko	Charkow	Recovered	30 wk. fetus, lived 1 hr.	By a letter to Sittner from the operator, dated July 12, 1902
July 23, 1899	Harry Lufkin	St. Paul, Minn.	Recovered	39 wk. fetus, deformed, lived 4 hrs.	*Am. J. Obst.*, 41: 289, 1900
Sept. 1899	E. B. Cragin	New York	Recovered	38 wk. fetus, lived 5½ mo.	*Am. J. Obst.*, 41: 740, 1900
Sept. 1899	C. Menge	Leipsiz	Recovered	32 wk. fetus, lived only a few minutes	*Zentralbl. f. Gynäk.*, 24: 25, 1900
Oct. 1899	P. Zweifel	Leipsiz	Recovered	22 wk. fetus, died during operation	2 Fälle von Extra-uterine Gavidität mit lebendem Kinde Diss., Leipsiz, 1901
1899	J. M. Black	Knoxville, Pa.	Died	12 mo. fetus, lived 8 hours (5000 gm.)	*Annals Gyn. & Ped.* (Jan.) 1900
Feb. 1900	Th. Landau	Berlin	Recovered	7 mo. fetus, lived 8 hours	Sittner: *Arch. f. Gynäk.*, 64: 526, 1901

Summary of Cases (*Continued*)

Date of Operation	Operator	Place of Operation	Result to Mother	Status of Fetus	Special Notes and Bibliography
Feb. 1900	Jakobs	Brussels	Recovered	38 wk. fetus, lived 29 days	*Bull. de la Soc. Belge de Gyn. et d'Obst.*, 11: No. 1, 1900
Mar. 3, 1900	A. Fischer	Tiflis	Died	40 wk. fetus, lived and "will live"	*Russki Wratsch*, No. 3, 1903
1900	Pryor	New York	Recovered	5½ mo. fetus, lived only a short time	By a letter from the operator to Sittner, dated June 21, 1902
June 1900	Cz. Stankiewicz	Warsaw	Recovered	35 wk. fetus, lived 2½ days	*Czas Lekarskie*, p. 465, 1900
July 18, 1900	E. F. Root	Salt Lake City	Died	38 wk. fetus, lived 4 days	*Am. J. Surg. & Gynec.*, 16: 204, 1902–03
Aug. 1900	P. Boryssowicz	Warsaw	Recovered	37 wk. fetus, lived 18 hrs.	By a letter to Sittner from the operator
Sept. 1900	H. Tuholske	St. Louis	Died	36 wk. fetus, lived	*Tr. South. Surg. & Gynec. Ass'n.*, 14: 38, 1901
Oct. 1900	E. B. Cragin	New York City	Recovered	Full term fetus, lived	*Am. J. Obst.*, 43: 96 (Nov.) 1900
Oct. 1900	Sneguireff	Moscow	Recovered	32 wk. fetus, lived	Letter to Sittner from Dr. Greife 1st Ass't. to Prof. Sneguireff
Oct. 6, 1900	Donald	Manchester	Recovered	21 wk. fetus, living at operation	*Trans. North of England Obs. & Gyn. Soc.* (Oct.) 1900
Nov. 1900	Handfield-Jones	London	Recovered	"Lives today"	Reply to an inquiry by Sittner
Jan. 27, 1901	E. Pasquali	Rome	Recovered	31 wk. fetus, lived 24 hrs.	U. Rolandi: *Policlinico* (Sez. practica), 1903
Apr. 6, 1901	T. N. and H. N. Rafferty	Robinson, Ill.	Died	40 wk. fetus, lived 13 hrs.	*Med. Record*, 60: 472, 1901
May 24, 1901	J. S. Ridell	Aberdeen	Recovered	22 wk. fetus. No note as to survival	*Brit. M. J.*, 12: 7, 1902
June 1901	W. Perlis	Kiew	Recovered	40 wk. fetus, lived but deformed	By a letter from the operator to Sittner, dated Aug. 6, 1902
Dec. 1901	A. Benckiser	Karlsruhe	Recovered	23 wk. fetus, presume it could not survive	By a letter from the operator to Sittner, dated May 2, 1906
Dec. 26, 1901	Lentz	Metz	Recovered	42 wk. fetus, lived 20 min.	M. Luxenburger: Zur Casuistik der vorgeschrittenen ektopischen Graviditaten bei lebendem Kind. Dissertation, Strassburg, 1902
Jan. 21, 1902	H. T. Inge	Mobile, Ala.	Recovered	29 wk. fetus, lived a few min.	*Ala. M. J.* (Sept.) 1902
Feb. 8, 1902	W. L. Estes	Bethlehem, Pa.	Died	?? wk. fetus, lived	*Penn. M. J.*, 6: 393, 1902
Apr. 26, 1902	Lindfors	Upsala	Died	27 wk. fetus, lived 4½ hrs.	Upsala Läk Förhandl, 1903
Apr. 1902	W. Perlis	Kiew	Recovered	40 wk. fetus, lived	By a letter from the operator to Sittner, dated Aug. 6, 1902
July 16, 1902	K. Pawlik	Prague	Recovered	26 wk. fetus, born living	G. Nový: *Časop. lék. česk.*, 1905
Aug. 8, 1902	A. Thompson	Odessa	Recovered	24 wk. fetus, lived a short time	*Sburn ak i sbens Boles*, p. 162, 1903
Oct. 4, 1902	A. Fischer	Tiflis	Recovered	40 wk. fetus, lived 2 yrs.	*Russk. Wratsch*, No. 3, 1903
Feb. 1903	Albertin	Lyon	Died	21 wk. fetus, born living	*Lyon méd.*, No. 52, 1903
May 27, 1903	R. Sauter	Lindau a. Bodensee	Recovered	22 wk. fetus, living ?	By a letter from the operator to Sittner, dated Apr. 23, 1906
June 12, 1903	G. Calderine	Bologna	Recovered	40 wk. fetus, lived	*Atti della Soc. italiana di Ost. e Gin.*, 4: (Oct.) 1903
July 17, 1903	H. Schmit	Linz	Recovered	37 wk. fetus, lived	K. Franze: *Wien. klin. Wchnschr.*, 17: 530, 1904
July 22, 1903	H. J. Paterson	London	Recovered	27 wk. fetus, lived a few mins.	*Trans. Obst. London*, vol. 47
Aug. 3, 1903	H. W. Freund	Strassburg	Recovered	9 mo. fetus, lived 6 hrs.	*Smml. Klin. Vortrag*, 448: 225, 1903–7
Sept. 11, 1903	Potocki	Paris	Recovered	38 wk. fetus, mentally defective, lived	*Rev. de Gynec. et de Chir. Abd. Paris*, 12: 362, 1908 or *Bull. méd. Paris*, 22: 55, 1908
Oct. 4, 1903	E. Pestalozza	Rome	Recovered	22 wk. fetus, living at op.	*Ginecologia*, Anno 1, Fasc. 2, 1904
Oct. 1903	R. Olshausen	Berlin	Died	42 wk. fetus, lived	F. Prüsmann: *Ztschr. f. Geburtsh. u. Gynäk.*, 52: 288, 1904
Nov. 4, 1903	A. Czyzewics jun.	Lemberg	Died	40 wk. fetus, lived	*Zentralbl. f. Gynäk.*, 28: 97, 1904
Nov. 17, 1903	R. Chrobak	Vienna	Recovered	40 wk. fetus, lived	H. Peham: *Wien. klin. Wchnschr.*, 17: 527, 1904
1903	v. Guéard	Düsseldorf	Died	56 wk. fetus, lived a few days	By letter from the operator to Sittner, dated Nov. 2, 1904
May 12, 1904	Diehl	.....	Died	11 mo. fetus, lived only a short time, normal	*Union Medicale du Nord-Est* (June 15) 1904

SUMMARY OF CASES (*Continued*)

Date of Operation	Operator	Place of Operation	Result to Mother	Status of Fetus	Special Notes and Bibliography
Sept. 12, 1904	C. S. Bonifield	Cincinnati	Recovered	37 wk. fetus, deformed, lived	*Cincinnati Lancet Clinic*, p. 66 (July 16) 1910
Sept. 1904	D. Popoff	St. Petersburg	Recovered	40 wk. fetus, lived 1 yr.	*Russk Wratsch*, No. 27–29, 1905
Nov. 4, 1904	Friedrich	Leipzig	Recovered	9 mo. fetus, lived	Gentsch: *Zentralbl. f. Gynäk.*, 51: 2440–43, 1924
?	Rosenberger	?	Recovered	Normal fetus, lived	*Gyogyaszat*, No. 51 (Dec. 23), 1906 (Not in Ac. Lib.)
Dec. 17, 1904	H. Spencer	London	Recovered	26 wk. fetus, lived a few minutes	By a letter from the operator to Sittner, dated June 3, 1906
Dec. 27, 1904	Potocki	Paris	Recovered	9 mo. fetus, lived, normal	Delcamp: Thèse de Paris, 1918
Jan. 3, 1905	Fournier	Amiens	Recovered	7 to 8 mo. fetus, lived ½ hour	*Bull. Soc. d'Obst. Paris*, 8: 6, 1905
Jan. 6, 1905	P. W. Michin	Charkow	Recovered	39 wk. fetus, lived 1 mo.	*Russk Wratsch*, No. 12, 1905
Jan. 27, 1905	v. Ott	St. Petersburg	Recovered	37 wk. fetus, lived	By a letter from Th. Landau to Sittner, dated May 20, 1906
Mar. 20, 1905	Th. Landau	Berlin	Recovered	40 wk. fetus, lived	A. Sittner: *Deutsche med. Wchnschr.*, 32: 1200, 1906
Mar. 26, 1905	J. A. Amann	Munich	Recovered	22 wk. fetus, living at operation	By a letter from Hörrmann to Sittner, dated Dec. 3, 1906.
July 7, 1905	R. Lumpe	Salzburg	Died	24 wk fetus, lived 2 hrs.	Th. Haagn: *Monatschr. f. Geburtsh. u. Gynäk.*, 22: 771, 1905
Sept. 12, 1905	J. Jerie	Prague	Recovered	43 wk. fetus, lived	*Časop. lék. česk.*, p. 913, 1906
Jan. 27, 1906	E. T. Hargrave	Norfolk, Va.	Recovered	9 mo. fetus, lived 1 hr.	*Va. M. Semi-Montbly*, 11: 317, 1907
Feb. 9, 1906	Adjaroff	Philippopel, Bulgaria	Recovered	40 wk. fetus, normal, lived	*Wien. klin. Wchnschr.*, 20: 1614, 1907
Apr. 19, 1906	Th. Landau	Berlin	Recovered	30 wk. fetus, lived	*Berl. klin. Wchnschr.*, 43: 1061, 1906
[Sittner ends]					
May 14, 1906	Allwood	Kingston, Jamaica	Recovered	8½ mo. fetus, lived 3 hrs.	*Brit. M. J.*, 1: 376, 1907
Nov. 9, 1906	Duff Lewis	Washington, D. C.	Recovered	8½ mo. fetus, deformed, lived 3 days (Pt. had eclampsia)	*Wash. M. Ann.*, 6: 315–20, 1907–8
Dec. 1, 1906	Geo. Lockett	Vancouver, B. C.	Recovered	9 mo. fetus, lived	*West Canada M. J.*, 2: 476, 1908
Apr. 10, 1907	Treub	Amsterdam	Recovered	7½ mo. fetus, lived	*Ann. de la Soc. Obst. de France*, p. 227, 1908
Apr. 24, 1907	Jacomet	Donai	Recovered	7½ mo. fetus, lived	*Eco méd. du nord.*, 11: 292 (June 23) 1907
July 23, 1907	Weiss	Schwerin i.M.	Recovered	9 mo. fetus, lived	*Zentralbl. f. Gynäk.*, 32: 251, 1908
Aug. 3, 1907	Conrad	Okla. City	Recovered	9 mo. fetus, lived	Blesh and Reed: *J. A. M. A.*, 1: 2137 (June 27), 1908.
Oct. 1907	Werder	.....	Recovered	8½ mo. fetus, deformed, lived only a short time	*Am. J. Obst.*, p. 796 (Nov.) 1908
Nov. 19, 1907	H. Maag	Naestved, Denmark	Recovered	9 mo. fetus, lived 4 hrs., deformed	Hornemann. *Zentralbl. f. Gynäk.*, 32: 668, 1908
May 24, 1908	Tissier & Wiart	Paris	Recovered	9 mo. fetus, lived, deformed	Tissier: *Le Bull. méd.*, 22: 600, 1908
Aug. 14, 1908	J. N. Warren	Sioux City, Iowa	Recovered	9 mo. fetus, normal, lived 10 hours	*New York State J. Med.*, 89: 894 (May 1) 1909
Nov 27, 1908	W. C. G. Kirchner	St. Louis	Recovered	9 mo. fetus, normal, lived. Ovarian pregnancy	*Trans. Am. Ass. Obs. & Gyn.*, 22: 189, 1910
	H. Greenberg	Milwaukee	Recovered	9 mo. fetus, living. Condition of fetus not given, nor is date of operation	*J. A. M. A.*, 542: 1517, 1910
Mar. 14, 1909	Willers	Odenburg Dornum (Ostfriesl)	Recovered	9 mo. fetus, lived	Essen: *Therap. d. Gegenw.*, 16: 360, 1914
Dec. 22, 1909	Hood	York, Eng.	Recovered	9 mo. fetus, lived	*Lancet*, 1: 1662, 1913
Apr. 2, 1910	Porter	Cincinnati, O.	Died	6 mo. fetus, lived a few min.	*Cincinnati Lancet Clinic*, p. 65 (July 16) 1910
June 9, 1910	Savage	Auckland, New Zealand	Recovered	Deformed fetus, lived, wt. 4 lbs. 10 ozs.	*Proc. Roy. Soc. Surg., London*, Gyn. and Obs. sec., 5: 72, 1911–12
Nov. 4, 1910	John Lane	Boston	Recovered	9 mo. fetus, lived 24 hrs.	*Boston M. & S. J.*, 164: 683, 1911
Mar. 29, 1911	I. M. Calloway	Shreveport, La.	Recovered	9 mo. fetus, lived	*New Orleans M. & S. J.*, 64: 641, 1912
Oct. 4, 1911	Schwartz	St. Louis	Recovered	9 mo. fetus, lived 10 10 hrs.	Q. U. Newell: *J. Missouri M. A.*, 17: 357 (Sept.) 1920

SUMMARY OF CASES (*Continued*)

Date of Operation	Operator	Place of Operation	Result to Mother	Status of Fetus	Special Notes and Bibliography
Oct. 28, 1911	Davis?	New York City	Died	Full term normal fetus, lived 18 hours	Published for the first time in this paper
Dec. 13, 1911	J. S. Horsley	Richmond, Va.	Recovered	9 mo. fetus, lived	*Surg. Gynec. Obst.*, 17: 58, 1913
July 19, 1912	A. Boquel	Angers	Recovered	7½ mo. fetus, lived	*Arch. Mens. d'Obst. et de Gynec.*, Paris, p. 277, 1912
Oct. 10, 1912	Schwartz	St. Louis	Recovered	9 mo. fetus, lived	Q. U. Newell: *J. Missouri M. A.*, 17: 357 (Sept.) 1920
Oct. 12, 1912	M. Plauchu	Lyon	Recovered	5 mo. fetus, deformed, lived only a short time	*Bull. Soc. d'Obst. et de Gynec.*, 1: 904, 1912
Jan. 11, 1913	Harper	Selma, Ala.	Recovered	9 mo. fetus, lived, normal, delivered a full term intra-uterine twin the day before. Intra-abd. twin weighed 2 ozs. more than intrauterine	Dubose: *Southern M. J.*, 8: 784, 1915
Mar. 28, 1913	Mariantschik	Babuschkin (Kiew)	Recovered	8 mo. fetus, lived 8 hrs., deformed	*Monatschr. f. Geburtsh. u. Gynäk.*, 90: 320–33, 1932
July 1, 1913	Fairbairn	London	Recovered	7½ mo. fetus, lived	*Proc. Roy. Soc. Med. London*, Sec. Obs. & Gyn., 12: 183, 1919
July 13, 1913	Beekman	St. Petersburg	Died	9 mo. fetus, lived 4 wks.	*Monatschr. f. Geburtsh. u. Gynäk.*, 40: 187, 1914
May 6, 1914	E. B. Cragin	N. Y. C.	Recovered	9 mo. fetus, lived	*Am. J. Obst.*, 70: 1019, 1914
Oct. 12, 1915	W. Geo. Lee	Chicago	Died	8 mo. fetus, lived ½ hour	*Surg. Gynec. Obst.*, 24: 317, 1917
Nov. 21, 1915	E. C. Moore	Los Angeles	Recovered	9 mo. fetus, lived, normal	*Surg. Gynec. Obst.*, 33: 65, 1921
Aug. 3, 1916	Vignard	Nantes	Recovered	7½ mo. fetus, deformed, lived	Mlle. Traverson: These de Paris, 1917
Sept. 27, 1916	Rader	Boyd Co., Ky.	?	9 mo. fetus, lived	*West Virginia M. J.*, 17: 465–473, 1923
Nov. 1916	Shockaert	?	Recovered	Almost 12 mo. fetus, lived a few hours	E. Zarate: *Rev. Fran. de Gyn. et d'Obst.*, 17: 625, 1922
Apr. 16, 1917	Devraigne & Lariboisiere	.....	Recovered	9 mo. fetus, deformed, lived	Delcamp These pour le Doctorat en Med., 1918, Contribution a l'etude Grossesse Ectop. avec enfant vivant, Paris, 1918
Apr. 25, 1917	Dorman	N. Y. C.	Recovered	9 mo. fetus, lived	*Am. J. Obst.*, 77: 134, 1918
Nov. 17, 1917	Dorman	N. Y. C.	Recovered	8½ mo. fetus, lived	*Am. J. Obst.*, 79: 782, 1919
1917	Ayers	Memphis	Died	Period of gestation not given, lived	Francher: *J. Tennessee M. A.*, 15: 279–85, 1922 (Discussion)
1917	McGhee	Memphis	Recovered	8 mo. fetus, lived 3 days	Francher: *J. Tennessee M. A.*, 15: 279–85, 1922 (Discussion)
June 18, 1918	Cowan & Montgomery	Danville, Va.	Recovered	9 mo. fetus, lived, normal	Cowan: *Kentucky M. J.*, 22: 538–54, 1924
Oct. 8, 1918	A. C. Beck	Brooklyn, N. Y.	Recovered	9 mo. normal fetus, lived	(Says 262 cases of living child after 5th mo. operated from 1809 to 1919.) *J. A. M. A.*, 73: 962–65 (Sept.) 1919
1918	Zweifel	Leipzig	Recovered	9 mo. fetus, lived	Gentsch: *Zentralbl. f. Gynäk.*, 48: 2440–43, 1924
Aug. 1920	?	New Orleans, La.	Died	9 mo. fetus, lived a few hours	*Am. J. Obst. & Gynec.*, 4: 71–75, 1922
Sept. 5, 1920	Nafe	Indianapolis	Recovered	9 mo. fetus, deformed, lived 1 hr.	*J. Indiana M. A.*, 19: 332–40, 1926
Sept. 20, 1920	Blackall	Fremantle, Australia	Recovered	9 mo. fetus, normal, lived	H. Stubbe: *Med. J. Australia*, 2: 570 (Dec. 25) 1920
Nov. 13, 1920	A. R. Rozer	Macon, Ga.	Recovered	9 mo. fetus, lived	*J. A. M. A.*, 76: 239 (Jan. 22) 1921
Feb. 22, 1921	Rowland	Baltimore, Md.	Recovered	9 mo. fetus, normal, lived	*Surg., Gynec. Obst.*, 42: 50–52, 1926
Apr. 25, 1921	Brugnatelli	Genua	Died	8 mo. fetus, lived	*Zentralbl. f. Gynäk.*, 46: 1831–36, 1922
Apr. 29, 1921	Nafe	Indianapolis	Died	9 mo. fetus, normal, lived 1 hr.	*J. Indiana M. A.*, 19: 332–40, 1926
June 1, 1921	O. Hoehne	Greifswald, Germany	Recovered	6 mo. fetus, heart beat, lived only a few mins., insufficient respiratory efforts, deformed	*Zentralbl. f. Gynäk.*, 47: 145, 1923
Oct. 1921	W. A. Jewett	Brooklyn, N. Y.	Recovered	8½ mo. fetus, lived a few hrs.	*Am. J. Obst. & Gynec.*, 5: 176–81, 1923
Nov. 8, 1921	E. O. Croft	Leeds	Recovered	8½ mo. fetus, lived 5 days	*Lancet*, 2: 380, 1922

SUMMARY OF CASES (*Continued*)

Date of Operation	Operator	Place of Operation	Result to Mother	Status of Fetus	Special Notes and Bibliography
Nov. 28, 1921	Dorman	N. Y. C.	Recovered	8½ mo. fetus, lived	*Am. J. Obst. & Gynec.*, 6: 219–21, 1923
1921	H. T. Hicks	Derbyshire	Recovered	9 mo. fetus, deformed, lived 30 hrs.	*Brit. M. J.*, 1: 141 (Jan. 28) 1922
Feb. 3, 1922	Guerin-Valmale & Robiolis	Marseille	Died	8½ mo. fetus, live	*Bull. Soc. d'obst. et de gynec. de Par.*, 11:538–42, 1922
Mar. 10, 1922	R. C. Van Etten	N. Y. C.	Recovered	9 mo. fetus, lived	*Am. J. Obst. & Gynec.*, 5: 206–07, 1923
Apr. 24, 1922	Rozas	Phill. Is.	Recovered	9 mo. fetus, lived	Rozas & Villarama: *J. Phillippine Islands M. A.*, 4: 50–54, 1924
June 29, 1922	O'Neill & Crawford	San D ego, Calif.	Recovered	9 mo. fetus, deformed, lived	*J. A. M. A.*, 80: 913, 1923
Aug. 8, 1922	O. Hoehne	Greifswald Germany	Recovered	9½ mo. fetus, not deformed, lived 15 hrs.	*Zentralbl. f. Gynäk.*, 47: 145, 1923
Sept. 3, 1922	Stephen	Greifswald Germany	Recovered	6 mo. fetus, showed signs of life for a few minutes	O. Hoehne: *Zentralbl. f. Gynäk.* 47: 145, 1923
Oct. 4, 1922	L. Rau	Far Rockaway, L. I.	Recovered	8½ mo. fetus, sl. deformed but healthy, lived	*Am. J. Obst. & Gynec.*, 5: 205–06, 1923
Jan. 9, 1923	Bermann	Buenos Aires	Recovered	9 mo. fetus, normal, lived only a few mins.	*Semana méd.*, 1: 1008–10, 1923
Jan. 18, 1923	C. H. Brown	Waterbury, Conn.	Recovered	9 mo. fetus, normal, lived	*Am. J. Obst. & Gynec.*, 7: 101–102 (Jan.) 1924
Jan. 19, 1923	Prof. Laurentie	Beyrouth	Recovered	5½ mo. fetus, deformed (made resp. movements a few minutes)	*Rev. franc. de gynéc. et d'obst.*, 18: 335–38, 1923
Apr. 12, 1923	Doderlein	Munich	Recovered	9 mo. fetus, lived 3 mo. (D. pn.)	H. Dehler: *Monatschr. f. Geburtsh. u. Gynäk.*, 68: 314–25 (Mar.) 1925
May 14, 1923	L. A. Ledoux	New Orleans	Died	? fetus, lived	*New Orleans M. & S. J.*, 76: 371–73 (Feb.) 1924
Feb. 20, 1924	Le Doux	New Orleans	Died	8½ mo. fetus, lived 1 hr.	*Am. J. Obst. & Gynec.*, 11: 395–99 (Mar.) 1926
June 17, 1924	Kwan	Canton, China	Recovered	7 to 7½ mo. fetus, lived 7 days	*China M. J.*, 40: 361–65, 1926
Oct. 8, 1924	Lacouture & Massé	Bordeaux	Recovered	9 mo. fetus, lived	*Bull. Soc. d'obst. et de gynéc. de Par.* 14: 294–8, 1925
?	Potter	Buffalo, N. Y.	Recovered	9 mo. fetus, lived, very sl. deformed	Hayd & Potter: *Am. J. Obst. & Gynec.*, 5: 601–20 (June) 1923
Mar. 13, 1925	P. S. Mills	Gaya, India	Recovered	Time of gestation not stated, lived	*Indian M. Gaz.*, 60: 510, 1925
Nov. 3, 1925	Beaucamp	Aschen	Recovered	9 mo. fetus, lived, normal	*Monatschr. f. Geburtsh. u. Gynäk.*, 76: 260–65 (June) 1927
May 23, 1926	Skinner	St. Paul, Min.	Recovered	? mo. fetus, sl. deformed, lived	*J. Am. Inst. Homeop.*, 21: 1043–47 (Dec.) 1928
Mar. 11, 1927	Recaséns	Madrid	Recovered	9 mo. fetus, normal, lived	V. Aza: *Arch. de Med. Ciry. Espec.*, 27: 489–93 (Oct. 29) 1927
?	Wm. P. Daly	?	?	?	*Am. J. Obst. & Gynec.*, 13: 649, 1927
Apr. 27, 1927	Williamson	Pittsburg	Died	9 mo. fetus, lived	*Am. J. Obst. & Gynec.*, 15: 215, 1928
May 8, 1927	McCullough	Philadelphia	Died	9 mo. fetus, lived	*Am. J. Obst. & Gynec.*, 17: 724, 1929
May 12, 1927	Micholitsch	Vienna	Recovered	9 mo. fetus, deformed, lived	*Zentralbl. f. Gynäk.*, 51: 2116–20, 1927
Nov. 13, 1927	Wagner & Hahn	Peru, Ind.	Recovered	9 mo. fetus, lived	*J. Indiana M. A.*, 21: 154–55, 1928
?	Masloff	Russia	Recovered	8 mo. fetus, normal, lived	*J. akusb. i zbensk. bolez.*, 39: 245–49, 1928 (Not in Academy Lib.) From the *Ber. ü. d. ges. Gynäk. u. Geburtsh.*, 15: 863, 1928–29
July 22, 1928	Mariantsch & Bychowski	Kiew	Recovered	10 mo. fetus, normal, lived	*Monatschr. f. Geburtsh. u. Gynäk.*, 90: 320–33, 1932
Sept. 28, 1928	Hayes	Melbourne, Australia	Recovered	9 mo. fetus, lived 24 hrs. (D. asphyxia neonatorum)	*Med. J. Australia*, 2: 17–18, 1933
Nov. 20, 1928	F. J. Brown	?	Recovered	9 mo. fetus, lived, normal	A. M. Richards: *Brit. M. J.*, 1: 807 (May 4) 1929
Feb. 23, 1929	Torrance & Bowman	?	Recovered	? fetus, deformed, lived	*Clin. Med. & Surg.*, 38: 483–85, 1931
Apr. 11, 1929	Gullichsen	Finland	Recovered	9 mo. fetus, lived ½ hr., club-foot	*Finska läk.-sällsk. handl.*, 71: 860–62, 1929

Summary of Cases (*Continued*)

Date of Operation	Operator	Place of Operation	Result to Mother	Status of Fetus	Special Notes and Bibliography
Apr. 17, 1929	Kenan Tevfik	Constantinople	Died	8 mo. fetus, deformed, lived 6 hours	*Zentralbl. f. Gynäk.*, 53: 2898–2902 (Nov. 16) 1929
July 30, 1929	Lash	Chicago, Ill.	Died	9 mo. fetus, lived 18 hours	*Illinois M. J.*, 65: 462–6, 1934
Aug. 1929	L. D. Wilcox	?	Recovered	9 mo. fetus, lived	*Canad. M. A. J.*, 23: 60–62, 1930
Oct. 11, 1929	Hangseth	Norway	Recovered	10 mo. fetus, lived	*Norsk mag. f. Laegevidensk*, 91: 304, 1930. From *Ber. ü. d. ges. Gynäk. u. Geburtsh.*, 18: 525, 1930–31
Oct. 13, 1929	Brindley	Temple, Tex.	Recovered	9 mo. fetus, normal, lived	*Texas J. Med.*, 26: 571–73, 1930
?	S. Takahashi & Y. Kitaura	?	Recovered	8 mo. fetus, lived 1 hr.	*J. Orient. Med.*, 13: 35 (Sept.) 1930
?	Froloff	Russia	Recovered	Lived	*J. akusb. i zbensk. bolez.*, 39: 448–51, 1928 (Not in Academy Lib.) From *Ber. ü. d. ges. Gynäk. u. Geburtsh.*, 17: 288, 1930
Jan. 23, 1930	A. H. Driver	Jammalamadugu Madras Presidency	Recovered	9 mo. fetus, "suckling at 4 days"	*Indian M. Gaz.*, 65: 273–74 (May) 1930
Jan. 29, 1930	Cornell	Chicago, Ill.	Recovered	8 mo. fetus, normal, lived	*Illinois M. J.*, 65: 462–6, 1934
May 30, 1930	A. W. Thrush	Chambersburg, Pa.	Died	8 mo. fetus, never breathed but born with heart beating. (Delivered per vagina)	*Pennsylvania M. J.*, 34: 37–38 (Oct.) 1930
Oct. 13, 1930	J. Mundell	Wash., D. C.	Recovered	9 mo. fetus, lived 3 hrs.	*Med. Annals Distr. Columbia*, 2: 86–90, 1933
Oct. 28, 1930	E. V. Caldwell	Huntsville, Ala.	Recovered	9 mo. fetus, lived	*South. M. J.*, 24: 729 (Aug.) 1931. (Says he finds only 122 cases like his in the literature, including his)
Oct. 29, 1930	Horner	Chicago, Ill.	Recovered	7 mo. fetus, normal, lived	Cornell & Lash: *Illinois M. J.*, 65: 462–66, 1934
?	Stratham & Shepherd	Bristol	Recovered	5 to 6 mo. fetus, lived 20 minutes	*Bristol Med.-Chir. J.*, 48: 15–34, 1931
About 1930	Pillai	Hongkong	Died	? mo. fetus, living. No note as to survival	Believed to have been a pregnancy in a rudimentary horn that ruptured early. Tottenham: *J. Obst. & Gynaec. Brit. Emp.*, 41: 56 (Feb.) 1934
Jan. 2, 1931	Lash	Chicago, Ill.	Recovered	8 mo. fetus, normal, lived	*Illinois M. J.*, 65: 462–66, 1934
Apr. 13, 1931	Spackman	Poona, India	?	Full term fetus, lived a few mins. Did not respire. Sl. deformity	*J. Obst. & Gynaec. Brit. Emp.*, 40: 1220 (Dec.) 1933
Feb. 24, 1932	F. Chanina	Gaiduk, Moscow	Recovered	9 mo. fetus, lived	*Monatschr. f. Geburtsh. u. Gynäk.*, 94: 22–28 (Apr.) 1933
Nov. 14, 1932	Geo. Nordlinger	Washington, D. C.	Recovered	7½ mo. fetus, normal, lived	First reported in this paper.
Nov. 26, 1932	Ware	Richmond, Va.	Recovered	8½ mo. fetus, normal, lived	*Am. J. Obst. & Gynec.*, 27: 756–9, 1934
1932 or 1933	Szlank	Krakow	Recovered	8 mo. fetus, deformed, lived 6 hours	*Ginek. polska*, 12: 30–37, 1933
1932	Raymond T. Holden	Washington, D. C.	Recovered	8 mo. fetus, lived a few hours	First reported in this paper
Feb. 16, 1933	Taylor	Durban, S. Africa	Recovered	9 mo. fetus, lived	*South African M. J.*, 7: 437, 1933
Feb. 22, 1933	Clemente	Palermo, Italy	Recovered	8 mo. fetus, born living but did not respire	*Ann. di ostet. e ginec.*, 55: 1479 (Sept. 30) 1933
May 16, 1933	Cruickshank	Tanjobe, India	Probably died	8 mo. fetus, normal, lived	Cruickshank & Achar: *Indian Med. Gaz.*, 69: 61–64 (Feb.) 1934
Sept. 5, 1933	Ziegler	Pittsburgh, Pa.	Recovered	9 mo. fetus, normal, lived	*J. A. M. A.*, 104: 2175–6, 1935
Sept. 10, 1933	A. C. Posner	New York City	Died	Full term fetus, still living, no deformity, perfectly well	First reported in this paper
Sept. 13, 1933	Alfred M. Hellman	New York City	Recoverd	Full term fetus, living, normal	First reported in this paper

A CLINICAL EVALUATION OF THE PATHOGENICITY OF TRICHOMONAS VAGINALIS*

ADOLPH JACOBY, M.D., F.A.C.S. AND MAURICE G. DERBRUCKE, M.D.

Assistant Professor of Gynecology, New York Post-Graduate Medical School, Columbia University

Instructor in Gynecology, New York Post-Graduate Medical School, Columbia University

NEW YORK CITY

IN recent years there have been many conflicting opinions regarding the role of trichomonas vaginalis in inflammatory conditions of the vagina. A review of the literature indicates that this is a repetition of the confusion following its initial discovery.

Donné[1] first called attention to this parasite in the vaginal secretion in 1837. In the subsequent twenty years it was looked upon as a simple ciliated cell, by Rokitansky,[2] Valentin and Vogel,[3] Gluze,[4] Seebald,[5] and Lebert.[6] Although mentioned by Felix Dujardin[7] in his "Infusorien," in 1841, by Leblond zu Brenner[8] and Robert Froriep,[9] it remained for Scanzoni and Kolliker[10] in 1856 to establish its positive identity. In a chapter devoted to the subject, Scanzoni described the trichomonas vaginalis, the vaginal discharge and the symptomatology, much as they are recognized today. He appreciated the importance of thinning the vaginal secretion for examination, which he did by adding water. Although first noticed in his pregnant cases, he found them in more than 50 per cent of both pregnant and non-pregnant patients. He likewise found them in "benign" as well as "virulent" vaginal discharges. He stated that the trichomonas vaginalis was not necessarily associated with gonorrhea, a premise originally advanced by Donne, who later admitted its fallacy.

Carl Hennig,[11] in 1870, credited Scanzoni and Kolliker with definitely establishing the trichomonas vaginalis as a unicellular parasite. He too contended that the trichomonas vaginalis "exists in the diseased increased mucus (discharge) of the cervical os and the upper portion of the vagina." He never observed them in or beyond the cervical canal. Then follow Hausmann,[12] 1870, Gasser,[13] 1874, and Künstler[14] 1884, all substantially agreeing with Scanzoni, Kolliker and Hennig. Massa[15] in 1885; Marchand,[16] 1894; Dock[17] 1895; N. S. Davis,[18] 1896; Mazzanti,[19] 1900; Brumpt,[20] 1913; Hoehne,[21] 1916, all considered the trichomonas pathogenic.

The American literature, in the main, has confined itself to verifying the claim of the pathogenicity of the trichomonad and outlining its treatment. Stein and Cope,[22] C. H. Davis,[23] P. B. Bland and his associates,[24] Cornell, Goodman, and Mattheis,[25] Furniss,[26] and Rowlett,[27] all consider this organism pathogenic. Hibbert,[28] Hesseltine,[29] and Hesseltine, Plass and Borts,[30] are some of the recent investigators who deny its pathogenicity.

In an attempt to decide the question, we considered the evidence furnished by the patient's symptoms, the associated objective findings, and the results of treatment. A routine examination of 300 consecutive, unselected cases was made. The only requisite, prior to examination for trichomonas, was that no douche or medication had been used during the preceding twenty-four hours. A fresh, warm, saline emulsion was examined for the flagellates.

* From the Department of Gynecology, New York Post-Graduate Medical School, Columbia University.

The ages of this group ranged from sixteen to seventy-two years. Fifty or 16⅔ per cent of these cases showed trichomonas vaginalis; 250 or 83⅓ per cent were negative.

TABLE I
LEUCORRHEAL DISCHARGE

Quantity			Color	
Cases Positive for Trichomonas (Per Cent)		Negative for Trichomonas (Per Cent)	Positive for Trichomonas (Per Cent)	Negative for Trichomonas (Per Cent)
Slight.....	29	70	White 45	55
Profuse.....	71	30	Yellow 55	45

Of the 50 positive cases, 8 or 16 per cent had no vaginal discharge, while 42, or 84 per cent, had a vaginal discharge of some kind. Twelve of the latter 42, or 29 per cent, had a slight discharge while 30 or 71 per cent had a moderate or profuse leucorrhea.

An analysis of the type of discharge shows that in only 19 or 45 per cent of the cases with discharge was it white in color, and in only 11 of the 19 was it abundant and frothy. In the remaining 23, the discharge was yellow in color, and only occasionally green or brown. In other words, in only 11 of the 50 positive cases was the discharge abundant, white and frothy, the type of discharge asserted to be characteristic of trichomonas invasion.

A comparison with the cases negative for trichomonas, showed that 136 had a discharge, of which 94 or 70 per cent were slight, and 42 or 30 per cent moderate or profuse. Of these 136, 75 or 55 per cent had a white, and 61 or 45 per cent had a predominantly yellow discharge. Of the 75 cases with a white discharge 16 were profuse and frothy. It appears therefore that a large proportion of these cases had a white discharge, with no trichomonas. It may be logically deduced that either a white frothy discharge is not characteristic of trichomonas, or trichomonas does not produce a white frothy discharge, or any other discharge.

TABLE II
ITCHING

Positive for Trichomonas		Negative for Trichomonas	
Discharge (Per Cent)	Itching (Per Cent)	Discharge (Per Cent)	Itching (Per Cent)
84	34	54	15.2

The analysis shows itching as a symptom to be directly proportional to the amount of discharge present. In the group positive for trichomonas vaginalis 17 or 34 per cent complained of itching, while only 38 or 15 per cent of the negative class had any itching. In conjunction with this finding, it is important to note that 30 of the 50 positive cases, or 60 per cent had a moderate to profuse discharge, while 42 of the 250 negative cases, or 16.8 per cent had a moderate or profuse discharge. It thus appears that the itching is proportional to the quantity of leucorrhoea.

TABLE III
VAGINAL INFLAMMATION

Number of Cases		Vaginal Inflammation	Per Cent
Positive.....	50	15	30
Negative.....	250	17	6.8

Vaginal inflammation of any degree was present in only 15 or 30 per cent of the trichomonad cases, and in 17 or 6.8 per cent of the others.

From the foregoing data it is evident:

1. That a large proportion of patients harboring the trichomonad manifest no symptoms of infection, such as milky discharge, itching or objective evidence of vaginal inflammation.

2. That a large number of patients with symptoms ascribed to trichomonas vaginalis, and with definite inflammation of the vaginal mucosa, show no trichomonads.

Of further significance is an evaluation of the treatment directed toward the

elimination of the trichomonas vaginalis. Similar confusion is evidenced in the methods of treatment advocated. Davis and Colwell[31] have shown that innumerable therapeutic agents will destroy the trichomonas vaginalis.

DeLee[32] in 1920 urged vigorous scrubbing of the vagina with tincture of green soap, irrigating with 1:5000 bichloride of mercury, and finally using a glycerine and sodium bicarbonate pack. Kane[33] anesthetized the patient, in order to scrub more thoroughly. Greenhill[34] now uses hexylresorcinol to swab the vagina, after a scrubbing sufficient to produce slight bleeding. Schmid and Kamniker[35] use a 0.1 per cent bichloride of mercury douche and a borax and glycerol swab and suppository. Faulkner[36] likewise scrubbed the vagina but used methylene blue as a swab, and boroglycerid tampon, followed by lactic acid douche. P. B. Bland and his associates, aside from the scrub, washed with 1 per cent cresol and later swabbed the vagina with an aqueous solution of 1 per cent picric acid, followed by a picric acid douche. H. S. Morgan[37] used a variety of methods. Following the scrubbing with soap, and drying the vagina, he blew in some antiseptic powder, which in turn was followed by a lactic acid douche. At other times he would paint the vagina with Lugol's solution, air dry it, and then use a dusting or drying powder and followed by methylene blue, potassium permangate, or a 1:4000 bichloride douche. Goodall[37] inserts a cone of picric acid and prescribes a soap or lactic acid douche. LeDoux[39] varied his treatment slightly. Following the scrub and bichloride douche, he swabbed the vagina with 2 per cent mercurochrome and added a tampon of zinc oxide or Lassar's paste. Graham[40] after the soap scrub used an iodine douche followed by an application of 5 per cent mercurochrome. C. J. Andrews[41] offers a complicated routine: tincture of green soap scrub; wash with sterile water; douche with 1:1000 bichloride of mercury; wash with sterile water; paint the vagina with 10 per cent silver nitrate; instill ½ ounce ichthyol and glycerine, protected by a rose-bud tampon. After removal the following morning; douche with ½ per cent lysol or cresolis. Repeat in forty-eight hours, then dry and blow in some antiseptic powder. Matters[42] uses the same scrub, but swabs with methylated spirits and 2 per cent acriflavine, followed by a glycerine and acriflavine suppository, and a douche the following day with either alum or lactic acid. Northrup[43] uses 1 per cent metaphen and soap for scrub. After drying, he paints the vagina with picric acid, dries it again, and packs it with powdered sulphur, which is held in place by a fluff of gauze. This is followed by a lactic acid douche. Cummings[44] was advised by Marshall, one of his colleagues, to use neosalvarsan intravenously, but contented himself with filling the vagina with 0.3 gm. neosalvarsan ointment. He does not scrub the vaginal tract, but simply washes the vulva with tincture of green soap. Kamperman[45] after the scrubbing contents himself with bathing the vulva with 50 per cent glycerine and a lactic acid douche. Purcel,[46] uses copper sulphate ionization and copper sulphate for a douche. Kleegman[47] after the scrub bathes the vagina in pyroligneous acid and packs with Lassar's paste. This she continues through the menstrual period. Gellhorn[48] resorts to the insufflation of acetarsone. The cinchona derivatives, which have been used by the earliest investigators are again brought into use. Rodecurt[49] uses Yatren 105, a cinchona compound and Sure and Bercey[50] reintroduced the use of quinine sulphate insufflation.

It is evident from the variety of medicaments advised that none of them is universally satisfactory. We have used at different times all of the treatments advocated, including the soap scrub, mercury douche, the dye paintings, the astringent packings, quinine insufflation and the various drying and antiseptic

powders. A method that afforded us some satisfaction was the application of oxyquinoline tartrate to the vaginal tract and the portio. We soon discarded the scrub and dissolved the oxyquinoloine tartrate by gently rubbing the tablet around the vaginal walls. A smaller lozenge-shaped tablet was inserted into the cervical os, and held there until it, likewise, began to dissolve. The subjoined tables show our results with the more popular agents.

TABLE IV
CASES DISCHARGED AS "CURED"

	Cured (Per Cent)	Failure (Per Cent)	Unknown (Per Cent)
Tr. green soap, scrub, mercury douche dye application	12.5	50	37.5
Quinine-sulphate insufflation, zinc chloride douche	45.4	36.3	18.3
Oxyquinoline tartrate dissolved in vagina and portio	57.14	42.86	0
Combination of local treatment and electrotherapy to cervix for eradication of pelvic pathology	91.6	8.4	0
No specific treatment	1 patient	1 patient	

Our observations extended over a period of many months, in some cases, years. There were periods of weeks, even months, when no parasite could be demonstrated. However, if a chronic pelvic inflammatory condition persisted, a chronic cervicitis or endocervicitis remained uncured, sooner or later the trichomonas would again be demonstrable. In our hands, the soap scrub, douche, dye treatment, yielded a 12.5 per cent cure and a 50 per cent failure. Quinine sulphate insufflation followed by zinc chloride douches on alternate days resulted in a 45.4 per cent cure and a 36.3 per cent failure. Using oxyquinoline tartrate 57 per cent were cured and 42.8 per cent were classed as failures. Two cases receiving a variety of treatments, including glycerine packs, mercurochrome packs, pyroligneous acid, etc. One of these remained cured.

The treatment that yielded the most consistently satisfactory results was the one that was directed less to the trichomonas vaginalis, and more to the eradication of the pelvic congestion, the cervicitis, and the endocervicitis. In this group the cervices were subjected to either actual electrocauterization, coagulation, or conization. The elimination of the pelvic pathology supplemented by any of the other treatments yielded a 91.6 per cent cure and 8.4 per cent failure.

Of further interest is a review of the late followup, six months to two years later, of these so-called cures. The percentages quoted include only those patients traced and re-examined.

TABLE V
LATE FOLLOW-UP OF "CURED" CASES

	Recurrence of T.V. and Absence of Symptoms (Per Cent)	Recurrence of Symptoms and Absence of T.V. (Per Cent)	Free from All Symptoms and T.V. (Per Cent)
Tr. green soap, scrub, mercury douche, dye application	12.5	....	12.5
Quinine-sulphate insufflation, zinc chloride douche	5	35	30
Oxyquinoline tartrate dissolved in vagina and portio	....	14.28	28.56
Combination of local and electrotherapy to cervix for eradication of pelvic pathology	0	16.6	75
No specific treatment			

Of the first group, those subjected to the soap scrub, douche, dye treatment, 12.5 per cent had a return of the trichomonas without a return of the symptoms, while 12.5 per cent remained parasite and symptom free.

Of the successful cases treated with quinine sulphate and zinc chloride, 5 per cent had a recurrence of the trichomonas without symptoms, 35 per cent had a recurrence of the symptoms without a return of the trichomonas, and 30 per cent remained permanently cured of both symptoms and parasites.

Of those treated with oxyquinoline tartrate 14.28 per cent had a return of the symptoms without a recurrence of the trichomonas, and 28.56 per cent remained symptom and parasite free.

The combination of electrotherapy and local treatment of the pelvic pathology showed that 16.6 per cent had a return of the symptoms without a return of the parasite, and 75 per cent remained symptom as well as protozoon free.

DISCUSSION

The trichomonas vaginalis is essentially a surface parasite. It can readily be washed away with a simple douche, as evidenced by the uniform efficiency of the washes and douches advised. The chronic congestive factor is not so easily removed, as evidenced by the great variety of depletives, germicides and drying agents used. The congested pelvic organs and the vagina furnish a fertile culture media for numerous bacteria, and the flagellates of the colon-typhoid group in particular. The simple eradication of the trichomonas vaginalis will not appreciably affect the symptoms for which the patient seeks relief. This is well demonstrated by the end-results in our cases. The parasite was repeatedly eliminated by several methods of treatment directed toward the trichomonad, but it frequently returned. A considerable number, regardless of the method of treatment, had a recurrence of the symptoms despite the absence of the parasite. Where the therapy was primarily directed toward the eradication of the pelvic inflammatory process and passive congestion, the parasite completely disappeared. Undoubtedly all of the methods advocated by the various investigators affected to some extent a concomitant inflammatory process, and to that extent eliminated the protozoa.

SUMMARY

Three hundred consecutive patients were examined for trichomonas vaginalis. Fifty or 16.6 per cent showed the presence of the parasite. Sixteen per cent of the positive cases had no discharge of any kind, 24 per cent had only a slight discharge, making a total of 40 per cent of the patients positive for the presence of trichomonas vaginalis, in whom the leucorrheal discharge was a negligible factor.

Only 34 per cent of the trichomonas vaginalis patients complained of itching and only 30 per cent had any evidence of a definite vaginitis. Certainly such figures do not establish a definite disease entity.

Various vigorous treatments, gathered from different parts of the world, including our own, showing some success in eliminating the trichomonas vaginalis, have been mentioned.

The treatment that yielded the best results, in our hands, was that one directed toward the elimination of the chronic, congestive condition in the pelvis and not the trichomonas. With the eradication of the causative factor of the stasis, all of the symptoms as well as trichomonads disappeared in 75 per cent of the cases, and the trichomonas vaginalis disappeared permanently in 91.6 per cent of those so treated.

CONCLUSIONS

1. The trichomonas vaginalis is a nonpathogenic, parasitic invader of the normal vagina or of an already diseased vaginal tract.

2. In its invasion of a normal vaginal canal no symptoms are produced.

3. In a diseased vaginal canal the symptoms are those of the disease present and are not initiated by the trichomonas.

4. Treatment directed toward the destruction of the trichomonas is unnecessary.

5. Effective treatment of the underlying inflammatory condition, cervical or vaginal, will cure the patient.

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CORRECTION OF PARALYTIC INSTABILITY OF THE PELVIS*

C. L. LOWMAN, M.D., F.A.C.S.

LOS ANGELES, CALIF.

AT the Toronto meeting of the American Orthopaedic Association in 1931, we first reported on 11 cases of operation for fascial repair of the paralyzed abdominal wall. During the past three years, constant observation of, and experience with the operation and its results, carried on by various members of the staff of the Orthopaedic Hospital in Los Angeles, have confirmed the usefulness of this procedure. At the same time, our study of the muscle imbalances affecting the security of the trunk has aided in establishing the importance of pelvic stability in relation to functional improvement in the use of the extremities.

Three major imbalances usually result from partial or total paralysis of the abdominal wall. First of all, there is obliquity of the pelvis on the hip joints, resulting in lumbar lordosis; second, lateral displacement of the trunk on the pelvis; third, rotatory displacement with malposition of the vertebral column.

In regard to the first: as in all other imbalances, the rotation of the pelvis forward on the bi-femoral axis is modified in degree by the amount of over-action of viable opponents to the abdominal wall musculature. When lower back muscles are intact or are stronger than the abdominals, the sacrum is drawn up and the lordotic curve is increased. When the hip joint flexors are activated, they pull the unstabilized lumbar spine forward, further increasing the lordosis. Likewise, the pelvis, being in a continuous state of flexion on the femurs (all hip-joint flexors shorten and tend to fixed contractures) constantly over-stretches the weakened abdominals.

All movements of the leg, in flexion, as in walking or leg raising, require directional control, which is accomplished by the laterally placed adductor-abductor groups and rotatory control by the internal-external rotators. Among the powerful adductor-internal rotator group of the thigh, the adductor longus plays the most conspicuous part.

Since it is attached high up on the symphysis pubes, it is directly opposed to the rectus abdominis. Whenever the leg is brought forward or raised, it acts as a synergist to the hip-joint flexors. Especially in controlling direction of the thigh movement during flexion is this adductor longus powerfully activated, pulling heavily on its attachment, of origin, the symphysis pubis. Hence, in the presence of abdominal paralysis, with this muscle intact, the weight of the leg drags downward on the symphysis, with a similar pull through the muscles attached to the anterior inferior and superior iliac spines, immediately rotating the pelvis forward on the heads of the femora into a position of marked forward inclination.

As to the second deviation: whenever the paralysis extends laterally to involve the quadratus lumborum, abdominal obliques and transversalis (with usually involvement of the erector spinae group more on one side than the other) all efforts at walking necessitate displacement of the trunk laterally, in order to draw these muscles taut, before the pelvis and the leg below it can be raised. The marked effort to obtain lateral stabilization activates the stronger, or less affected muscles on one side, which means that the trunk is

* Read before the Pan American Medical Congress.

pulled over to take up the slack on the opposite weak side and thus puts a very heavy stretch on these weaker muscles. This added stretch paresis is an important factor in the progressive tendency towards lateral spinal deformity. The sag down on the weak side and the pull up on the strong produces a pelvic deviation which gives the relative effect of having a longer leg on the weak side. So much more effort is required to get the foot off the floor on this side that the pelvic deviation and spinal deformity increase constantly as soon as the walking stage is reached.

The third or rotatory deviation: As we brought out in the paper read at Toronto there is a very definite causative relationship between abdominal paralysis and the rotary deformation of paralytic scoliosis. The abdominal muscles are among the most important of the trunk-twisting muscle groups that rotate the trunk on its long axis on the fixed pelvis, or the whole body on the feet, when fixed by body weight on the ground.

For example: to reach forward with your right arm, you activate antero-laterally on the right side from below upward, chiefly the lower left abdominal obliques, rectus abdominis, right transversalis, right external oblique, right serratus magnus and right rhomboideus. At the same time, postero-laterally on the left side, from above downward, you activate left trapezius, left rhomboideus, left latissimus dorsi, left quadratus lumborum, left lower sacrospinalis and the intrinsic small spinal rotators.

In turning, more or less on a central vertical spinal axis, the rectus abdominis acts as a fixator or stabilizer. To it, the obliques and the transverse abdominals are inserted. If the lower left oblique, upper right oblique, upper or lower halves of the rectus abdominis are (any or all of them) partially or wholly incapacitated, the fixation of the right costal margin is insufficient. The right serratus magnus, which depends on these muscles to hold a fixed base, will, in the presence of their paralytic involvement, tend to flare the ribs outward and backward on that side. If the right rhomboideus is weakened by paralytic involvement, or stretched through simple postural relaxation, the right shoulder will be drawn forward, but the action of trunk torsion will be incomplete and not correctly controlled. Because of approximation of origin to insertion, contracture of the right serratus magnus takes place, the pectorals and gravity hold the weight mass of the arm and scapula against the side of the thorax, anterior to the rib angle, thus flattening the ribs and tending to increase the dorsal scoliotic deformity.

It may then readily be seen that paresis or paralysis of various extrinsic stabilizing muscles of the trunk greatly modify arm or leg functioning, since in order to obtain a stable foundation from which the extremities work, all the slack in the stretched and weakened muscles must be taken up before action can take place.

This fact was brought home to us very strikingly in the second case of fascial repair of abdominal wall upon which we operated. This patient had a paralysis of the upper abdominals and a moderately involved right rhomboideus. It was recorded that the inability to raise the left leg through a full arc was caused by a 50 per cent weakness of the left thigh flexors.

After fascial extensions were made, uniting the lower healthy rectus abdominis with the costal margin, one of the patient's earliest improvements was her ability to raise easily the extended left leg through a complete arc, in the lying position. This proved that the original diagnosis of paralysis of the left psoas magnus was incorrect, and that as an intrinsic flexor, it could not function properly in the presence of the loss of pelvic stabilization on the thorax, due to absence of the upper rectus abdominis. Prior to operation, the effort to raise the leg immediately rocked the pelvis downward, due to the lack of fixity of the pelvic position, hence the leg muscles only had sufficient purchase to raise the leg a short way.

The well-known presence of the movement of the umbilicus towards the unparalyzed portion of the abdominal muscles, made during efforts to raise the head, the legs or upper trunk, is definite evidence of paralysis or paresis. The marked interference in such actions as raising the head, turning the body, reaching forward, leaning the body forward from the back of a chair, or raising the pelvis and legs, as in walking, are all indications of the disturbance of the stabilizing function of the abdominal wall musculature.

To date, 23 of these operations have been done at our clinic by five different operators, with uniformly good results. These are confirmed by statements of the patients and their parents, by observations made by the different doctors and by records in kinesiological charting of the physical therapy technicians.

A very important result of this fascial repair operation has been the change for the better in the general health and well being of the patients operated upon. This, we consider largely due to the restoration of abdominal wall resistance to internal pressure. The improved support of the viscera, as well as the restoration of resistance to the downward force of the diaphragm in breathing, is strikingly shown in the following case which I am now presenting for the first time.

This case is the first application of the fascial repair of an abdominal wall with a lower body paralysis due to a crushed spinal cord, caused by fracture of the vertebral column.

On June 7, 1931, in an automobile accident, a woman of forty years sustained a fracture of the spine with complete paralysis below the level of the umbilicus, involving bladder and bowel. Four months later we started her treatment at the Orthopaedic Hospital in Los Angeles. She was given remedial reeducational exercises in the under water gymnasium as well as massage treatments in our private gymnasium.

Some improvement in the lateral trunk muscles suggested further possible improvement by laminectomy, and this operation was performed. The cord was found to be flattened to a few millimeters thickness and angulated over a ridge at the level of the fracture.

Some months later both adductors were tenotomized because of the very annoying spasticity of the adductors with the tendency to scissor-leg deviation.

We finally were able to get the patient on her feet in leg braces and a back brace, so that she could hold to upright supports. After some time, the left quadratus lumborum returned to sufficient strength to lift the left side so that the patient could advance the left leg. The right side could not compete with the strong overaction of the left quadratus, even though it too had some returning power. A marked upward movement of the umbilicus took place upon any effort to perform any movement. Without a back brace, the patient could not hold up her head, turn her body, nor sit up for any length of time.

At operation, on April 18, 1933, we implanted two fascial straps, 2 cm. wide, under the aponeurosis of each rectus abdominis, from a point 2 inches above the umbilicus down to the symphysis. Another strip of similar width was run obliquely downward from the same attachment on the upper rectus, but instead of attaching it to the right ilium, as we had been doing in the poliomyelitis cases, it was carried down to the leg and attached to sartorius and tensor fascia femoris, whose origin at the anterior spine of the ilium had been detached including a small button of bone and carried inward into line with the fascial insert. We felt sure that this would tend to lift up the sagging right side and that it might actually transmit some power to flex the hip joint enough to advance the leg.

Our reasoning proved to be correct, and at this time the patient has progressed so that she is able to walk forward and backward in a walker, and now makes as good a step forward with the right leg as with the left, sometimes better. Her ability to balance herself has greatly improved.

The most notable improvement, however, is the return of stability to the pelvis and trunk. She can now raise and hold up her head while recumbent, reach forward and push or pull with either hand, turn over completely in the water and partly so in bed, sit forward in her chair, etc. All of this she could not do before.

She states that her body feels taut and firm, as it did before the accident, and its general tone has markedly improved. The bladder condition has improved, chiefly because there is now enough resistance to downward pressure of the diaphragm during evacuation, so that the residual in the bladder is greatly lessened. Likewise, the increased ability to turn over, twist and bend has prevented the precipitation of mucus and sediment in the bladder, causing a lessened amount of cystitis; her analyses too are better than before operation.

This patient is now able to sit comfortably for hours at a time in a chair or automobile. She can also operate a typewriter with but a normal amount of fatigue. Recently, her back brace was replaced by a corset, as by wearing it she obtains better lateral sway in walking, and she no longer needs the rigid support of the stiff brace. Her general posture is good and her morale is marvelously improved.

CLINICAL OBSERVATIONS

A questionnaire to ten orthopedic surgeons and seven technicians elicited the following information.

1. Five surgeons had operated on 23 cases.

2. All agreed the results justified the operation.

3. All agreed and some emphatically, that abdominal muscle paralysis affects pelvic and spinal alignment.

4. All agreed that spinal fusion without thoraco-pelvic stabilization is not sufficient to maintain trunk fixation.

5. In regard to sequence, all stated it varied with the case as to whether fusion or fascial operation was done first.

6. All stated that physiological as well as structural improvement occurred after this operation, within a very short time.

7. No failures or deaths were reported.

8. As to placing fascial bands from pelvis to ribs in complete paralysis in girls, all agreed it was justifiable, recommending sterilization.

LIPOMA OF THE RECTUM

WITH REPORT OF A CASE

SYDNEY D. WEINSTEIN, M.D. AND WILLIAM LIEBERMAN, M.D.

BROOKLYN, N. Y.

THIS case is reported because of the rarity of lipoma of the rectum. Fatty tumors or lipomas were discovered by Alexis Littré, the anatomist, in 1704. They may occur almost anywhere in the body and have been reported in such diverse localities as the femoral vein, the adrenals, brain, orbit, pia mater, muscles, skin and almost every other organ. In the gastrointestinal tract they are of two varieties, submucous and subserous, depending upon whether the growth occurs under the mucosa or serosa, respectively. Cruveilhier, 1791–1874, appears to have been the first to mention submucous lipoma. Either type, although of a benign nature, may be malignant by virtue of growing to a size sufficient to occlude or compress the alimentary canal, producing obstruction. In this paper only the submucous type of lipoma of the rectum is considered, i.e. the truly intra-intestinal type, within the lumen of the rectum, which is the more important form. It is to be noted that the greater portion of the rectum is devoid of serosa. The place of lipoma among the benign tumors of the rectum is shown in the following classification:

Adenoma
Polyp
Villous tumor
} All are types of adenoma, named according to morphology.

Embryological defects: Teratoma
Dermoid

Lipoma: Submucous
Subserous
In ischiorectal fossa or hollow of the sacrum.

Fibroma, myoma, chordoma, anterior sacral meningocele, endometrioma (in vaginal septum), angioma, inflammatory tumors, myxoma, lymphoma, etc.

The structure of a rectal lipoma is the same as that of lipoma elsewhere, that is, normal fatty tissue in lobules held together by connective tissue trabeculae. If the fibrous tissue is greater in quantity it is called a fibro lipoma. The vascular supply is small. The mucosa covering it may be normal or ulcerated. Calcium salts may be deposited within the tumor. It is slow growing and never undergoes malignant changes.

THEORIES OF FORMATION[20]

Cicala thinks that the perivascular infiltration in the connective tissue trabeculae represents points from which the tumor grew, and that these cells changed from simple connective tissue cells to adipose tissue. Borst considers them the result of metaplasia of connective tissue into adipose tissue. Minelli holds that they represent the output of degenerated connective tissue, with new formation of fibroblasts, some of which make normal connective tissue, while others degenerate into fat. Many others think inflammation has some relationship. L'Aievoli places the responsibility on faulty lymph-blood circulation. Vaccari thinks neither trauma, infectious diseases, tuberculosis, alcohol, smoking, nor arteriosclerosis can explain origin of these tumors.

INCIDENCE

In the gastrointestinal tract malignant tumors are found more frequently than are the benign types.[14] Of the benign tumors, the most common are adenomas and polyps, with lipoma next in frequency. Lipoma of the gastrointestinal tract is of moderate rarity; lipoma in the rectum is very rare.

To give an impression of the degree of rareness, and the distribution of gastrointestinal lipomas, we cite the figures of various authors who have made exhaustive searches of the literature: Dewis[18] in 1906 was able to gather records of 6 cases of rectal lipoma. Stetten[17] in 1909 listed 72 cases of gastrointestinal lipoma of which 7 were rectal. Comfort[14] in 1931 reported 96 additional cases of gastrointestinal lipoma plus 13 culled from the literature, bringing the total to 181 cases. This added 3 more rectal cases, one which he reports himself, and 2 reported elsewhere.

Finally Poston[15] collected 242 recorded cases of lipoma in the entire gastrointestinal tract from a review of the literature of the world up to 1934. Of these only 11 were rectal, although benign new growths are said to occur five times as often in the rectum as in the small intestines and colon. This is surprising in view of the small area provided by the rectum in comparison with that of the colon and small intestines. Possibly ease of diagnosis in the rectum may account for it. But from the foregoing statistics it is seen that lipoma is an exception, only 11 cases being given as rectal. The reason for this may provide an interesting speculation.

By a careful search to exclude overlapping enumerations, we have found a total of 13 cases of rectal lipoma, 2 of which however are in some doubt as to the location of the growth. The 13 cases are reported by the following: Castelain[1] 1870, quoted by Molliere;[2] Afezou[3] 1875; Vois[4] 1880 (doubtful), quoted by Hiller;[5] Tuffier[6] (doubtful); Tedenat[7] 1885; LaGarde[8] 1886; Bodenhamer[9] 1898; Gant[10] 1902; Tuttle[11] 1902, 2 cases; Vogeler[12] 1924; Partsch[13] 1924; Comfort[14] 1931.

If we omit the 2 doubtful cases (in which the tumors were felt in the rectum but intussuscepted sigmoid was present to which it is probable the tumor was really attached) and add our case, we find a total of 12 authenticated cases of rectal lipoma. Molliere also quotes another case by hearsay, which is not included in the present list.

PREVIOUSLY REPORTED CASES

It will be interesting to compare the findings and course in the previously reported cases.

1. Castelain's case:[1] Produced varying degrees of obstruction, until finally the tumor was passed spontaneously. Size: 12 × 6 cm., ovoid. Pedicle 2–3 cm. long. In the other case mentioned by Molliere[2] the tumor was also passed spontaneously. This was a hospital case on the service of Dr. Claude Bernard in 1874. The patient complained of constipation and a sense of weight in the rectum. The mass could be felt with the finger. It was passed spontaneously. We do not include this as one of the listed cases since the actual location of the growth appears not to have been definitely determined.

2. Afezou's case:[3] Aged eighty-three. Symptoms: colic, diarrhea alternating with constipation. Passed spontaneously. Recovered.

The Tuffier case[6] may be mentioned in passing as illustrating a serious danger in this condition. Here the tumor was palpated in the lower part of the rectum at the head of an invaginated segment of sigmoid. Removal resulted in death by peritonitis, apparently due to perforation of the sigmoid. The Vois case,[4] occasionally and erroneously referred to as a rectal lipoma, consisted of a mass the size of a goose egg which presented in the rectum at the head of invaginated gut (either sigmoid or upper rectum). It was removed and the invagination reduced by water pressure.

3. Tedenat's case:[7] Aged sixty-four. Size 13 × 6 cm. Pedicle 3 cm. Complained of digestive disturbances, constipation, diarrhea, blood and mucus in the stools. Removed through the anus. Recovered.

4. LaGarde's case:[8] Aged sixty-five. Located in upper rectum. Size 2½ × 1¼ inches. Pedicle 8 inches. Constipation,

mucus and blood in the stools. Removed through the anus. Recovered.

5. Bodenhamer's case:[9] Aged thirty-seven. Size of hen's egg. Removed through the anus. Recovered.

6. Gant's case:[10] Located 4 inches above anus. Size of walnut. Pedicle 1 inch long. Removed through the anus. Recovered.

7. and 8. Tuttle's cases:[11] Both removed through the anus, and followed by recovery.

9. Vogeler's case:[12] Severe constipation, pain on defecation, followed by vomiting. The tumor was 10 cm. in diameter, bled easily, was located just above the anus. Four days later an infarcted fibrolipoma was passed spontaneously.

10. Partsch's case:[13] The patient forcibly extruded the mass. It replaced itself spontaneously but recurred, and this time was irreducible. The pedicle attached 8 cm. up on the right lateral wall was ligated and cut with the cautery. The patient recovered.

11. Comfort's report:[14] Patient aged eighty-one. Lipoma 1 cm. in diameter was accidently found in the rectum at autopsy.

12. AUTHOR'S CASE

B. Z., male, aged sixty-one, baker, came to our office on March 6, 1931 with the following history. He was always regular until six months ago when he became constipated. On the morning of March 6, a mass came out through his anus and remained out. He bled and suffered pain. There was no loss of weight or feeling of weakness. There was no sense of fullness in the pelvis.

Examination revealed a lobulated mass, 7 × 4 cm., yellowish in color, covered by a layer of thin mucosa which was ulcerated at one point. The mass was attached to the lower end of the rectal ampulla posteriorly by a pedicle 6 cm. long.

Under 2 per cent novocaine anesthesia, the sphincter was dilated; the pedicle was carefully ligated and cut with the endotherm knife.

The pathological report was lipoma.

The patient made an uneventful recovery.

From these cases we derive these symptoms of lipoma of the rectum, in the order of their importance:

1. Varying degrees of constipation and obstruction.

2. Diarrhea alternating with constipation.

3. A sense of weight and fullness in the pelvis.

4. Pain on defecation.

5. Digestive disturbances.

6. Mucus and blood in the stool due to torsion of the pedicle or ulceration of the overlying mucosa.

Any or all of these may be present. The symptoms are not distinctive.

Examination may reveal a polypoid mass which sometimes can be forcibly extruded by the patient. The finger may reach it within the rectum and palpate a soft doughy mass, pedunculated, and possibly lobulated. Inspection through a proctoscope may reveal the yellowish fat shining through the thin mucosa covering the tumor. The doughy consistency and the yellowish color may hint at the nature of the growth but usually the diagnosis would not be made until after biopsy or excision, with the tumor cut open or examined microscopically. Diagnosis preoperatively is more possible in the rectum than at any other location in the gastrointestinal tract. Other suggestive factors to note in making the diagnosis are obesity of the patient, presence of lipomata elsewhere and the benign character of the growth.

From examination of the listed cases, we find the age of occurrence to vary from thirty-seven to eighty-three; and the incidence is about equal between the sexes. It is to be kept in mind in making a differential diagnosis, that the occurrence is in the so-called cancer age.

The complications are rectal obstruction, ulceration of the tumor mucosa with loss of blood, twisting of pedicle with necrosis of tumor, and prolapse of the rectum by pulsion on the pedicle.

The treatment is excision per rectum. The pedicle should be carefully ligated or even doubly ligated before it is cut.

If the tumor is sessile the mucosa may be split and the growth shelled out. A source of danger to be noted especially in cases where the pedicle is attached 4 or more inches above the anus on the anterior wall, is that of invagination of the peritoneum into the pedicle by pulsion upon the tumor during the process of defecation. When the pedicle is cut the peritoneum is apt to be opened and a fatal peritonitis may result. Pedicles in this location should not be cut too close to the bowel wall and should first be carefully and tightly double-ligated. The scalpel should be replaced by the endotherm knife.

CONCLUSIONS

1. Lipoma of the rectum is rare, only 12 authenticated cases, including ours, being reported.

2. The symptoms are not distinctive. The principal ones are constipation and a sense of weight in the pelvis.

3. The treatment is surgical.

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ARTIFICIAL FEVER THERAPY OF GONORRHEAL ARTHRITIS

REPORT OF THIRTY-ONE CASES*

H. WORLEY KENDELL, M.D.
Assistant Director, Department of Fever Therapy Research

WALTER W. WEBB, M.D.
Resident Physician in Medicine

AND

WALTER M. SIMPSON, M.D.†

Director of Diagnostic Laboratories and Department of Fever Therapy Research
Miami Valley Hospital

DAYTON, OHIO

ONE of the most frequent and disabling complications of gonorrhea is gonorrheal arthritis. In a high proportion of cases permanent deformity and disability, affecting one or several joints, is the end result. The fact that so many different methods of treatment have been employed provides evidence of the inadequacy of most or all of them.

During the past three years, the outlook for patients with gonorrheal arthritis has been enormously improved.

In March, 1932, a thirty-nine year old man was referred to us for artificial fever therapy because of resistant seropositive syphilis. The history and physical examination revealed that the patient also had active chronic gonorrheal arthritis of five months' duration, involving the right wrist. Gram-negative intracellular diplococci were found in large numbers in urethral smears. After the third artificial fever treatment, each of which consisted of five hours of fever above 105°F. (40.6°C.) at intervals of one week, all evidence of active gonorrheal arthritis had disappeared. The joint function, which had been practically nil, was restored to 90 per cent of normal. The urethral smears became negative for gonococci after the fourth treatment and have remained negative since that time.

Encouraged by this coincidental observation we decided to treat other cases of gonorrheal arthritis. We have now completed the course of fever therapy of 31 patients with gonorrheal arthritis.

Scattered reports[1–9] of similar favorable experiences with artificial fever therapy by physical methods have appeared in medical literature since 1932. The results obtained with a variety of physical modalities have been summarized and tabulated in a recent communication by Hench, Slocumb and Popp.[10] Of 33 cases mentioned in the 9 reports reviewed by these authors, data on the results obtained in the treatment of 24 patients could be calculated. Of these 24 patients, 22 (92 per cent) were "completely relieved" or "cured." Failure resulted in only 2 cases (8 per cent) and was attributed to inadequate fever production. Hench, Slocumb and Popp have treated

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† With the collaboration of Charles F. Kettering, SC.D., Fred K. Kislig, M.D. (deceased), Edwin C. Sittler, B.S. in Engineering, L. G. Kauffman, M.D., Jerome K. Hartman, M.D., Eunice Fraizer, M.A., Florence Storck, R.N., Mary Louise Wolff, R.N., Ruth Walter, R.N. and Florence Buroker, R.N.

16 patients suffering from gonorrheal arthritis with artificial fever therapy, utilizing the Kettering hypertherm. Nine of the patients had acute arthritis, while 7 had the disease in a chronic form. Eighty-eight per cent of the patients with acute gonorrheal arthritis were promptly "cured," or practically so, while the remaining 12 per cent obtained marked improvement. Of the 7 patients with chronic gonorrheal arthritis, all but one were markedly improved. The authors state: "In gonorrheal arthritis, results are so striking and apparently so superior to those obtained by other methods, that we can prescribe fever therapy as the method of choice with considerable assurance."

APPARATUS

At the beginning of this undertaking, several physical modalities were employed, including hot baths, cabinets heated with carbon-filament electric lamps, electric blankets, diathermy and radiothermy. The clinical results obtained with any of these methods were essentially similar, when the fever could be maintained at a sufficiently high level for a sufficiently long interval. The recognition of the fact that some of these methods possessed inherent hazards prompted us to attempt the development of a relatively safe and simple method for fever induction and maintenance.

The apparatus now used for fever induction and maintenance is an air-conditioned cabinet developed at the Miami Valley Hospital and at the Research Laboratories of the Frigidaire Division of the General Motors Corporation, Dayton, with the collaboration of Mr. Charles F. Kettering and Mr. Edwin C. Sittler. The name "Kettering hypertherm" has been applied to the apparatus. In the present stage of its development, the Kettering hypertherm* (Fig. 1) consists of an insulated cabinet in which the nude patient lies, with his head extending outside the cabinet. Sponge-rubber insulation is utilized in the neck region to permit the patient to shift his position. The patient lies on a comfortable air mattress, supported by a box-like bed, which is rolled in and out of the cabinet at will. In the rear of the cabinet is a small insulated fire-proof compartment in which the air-conditioning apparatus is housed. The dry-bulb air temperature is controlled by a thermostat. The wet-bulb temperature, which governs the percentage of relative humidity, is controlled by a humidistat or by a wet-bulb thermostat. The air-velocity within the cabinet is

Fig. 1. Kettering hypertherm.

* Fifty-five of these units have been lent to 20 medical research centers, strictly for investigative purposes. The physicians and nurses charged with this undertaking received special training in the Department of Fever Therapy Research at the Miami Valley Hospital before the apparatus was released. A simpler, smaller and less costly apparatus is now being developed. It is probable that this apparatus will ultimately be available to certain qualified institutions. Adequate preliminary training of physician- and nurse-personnel is an essential requirement for this type of work. Artificial fever therapy should be restricted to qualified institutions. Otherwise, this method of therapy is almost certainly doomed to a period of discredit, not unlike that which followed the introduction of roentgen rays. The production of artificial fever at a high temperature level is not adaptable to ordinary office practice.

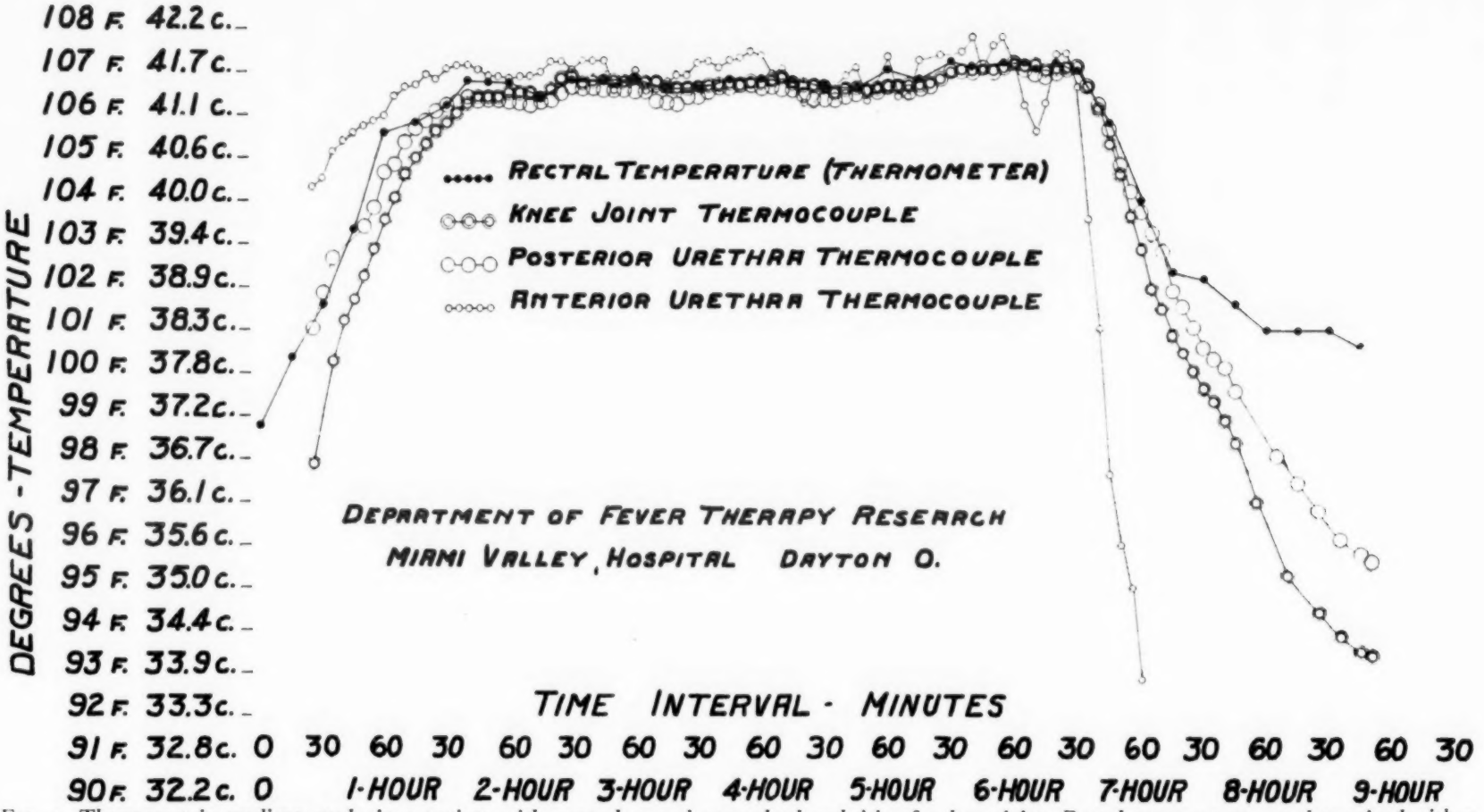
108 F. 42.2 c.
107 F. 41.7 c.
106 F. 41.1 c.
105 F. 40.6 c.
104 F. 40.0 c.
103 F. 39.4 c.
102 F. 38.9 c.
101 F. 38.3 c.
100 F. 37.8 c.
99 F. 37.2 c.
98 F. 36.7 c.
97 F. 36.1 c.
96 F. 35.6 c.
95 F. 35.0 c.
94 F. 34.4 c.
93 F. 33.9 c.
92 F. 33.3 c.
91 F. 32.8 c.
90 F. 32.2 c.
DEGREES - TEMPERATURE
RECTAL TEMPERATURE (THERMOMETER)
KNEE JOINT THERMOCOUPLE
POSTERIOR URETHRA THERMOCOUPLE
ANTERIOR URETHRA THERMOCOUPLE
DEPARTMENT OF FEVER THERAPY RESEARCH
MIAMI VALLEY HOSPITAL DAYTON O.
TIME INTERVAL - MINUTES
0 30 60 30 60 30 60 30 60 30 60 30 60 30 60 30 60 30 60 30
0 1-HOUR 2-HOUR 3-HOUR 4-HOUR 5-HOUR 6-HOUR 7-HOUR 8-HOUR 9-HOUR

FIG. 2. Thermometric gradient study in a patient with gonorrhea and gonorrheal arthritis of a knee joint. Rectal temperature was determined with a certified mercury thermometer at intervals of approximately fifteen minutes for nine hours. Temperatures of knee joint, anterior urethra and posterior urethra were determined with thermocouples at intervals of five minutes. Rectal temperature was elevated to 105.6°F. in one hour. Temperatures of various tissues reached practically same level as rectal temperature during maintenance period, with exception of temperature of anterior urethra which was slightly higher during induction and maintenance period, and exhibited fluctuations whenever skin surface of patient was cooled. Thermocouple temperatures were not recorded until twenty-five minutes after treatment was begun, due to mechanical difficulties. When rectal temperature had dropped to 100.5°F., posterior urethra temperature had dropped to 95.5°F.; knee joint temperature had dropped to 93.4°F.; and anterior urethra temperature had dropped to 92.8°F.

controlled by blowers of fixed speed. Dry-bulb and wet-bulb temperatures within the cabinet are indicated on large dials, equipped with warning pilot lights, on the top of the front end of the cabinet, where they may be constantly observed by the nurse-technician. The temperature-humidity factors may be controlled by the turning of a single knob. The average set of air conditions to which the patient's body is subjected is: dry-bulb air temperature of 140°–150°F. (60°–65°C.), relative humidity of 35–40 per cent, and air velocity of 425 cubic feet per minute. The elevation of the rectal temperature to 105°F. (40.6°C.) is ordinarily accomplished in forty minutes to one hour. The air is constantly conditioned by continuous passage through the air-conditioning compartment. The safety and comfort of the patient are greatly enhanced by the accurate control of the relative humidity.

The mechanism of fever induction with the Kettering hypertherm depends primarily upon heat transfer by conduction from the circulating heated air and the heated air-mattress. This factor, combined with prevention of the normal rate of heat loss from the body by radiation and evaporation, is responsible for the elevation of the body temperature and its maintenance at any desired level.

The simplification of the apparatus, the removal of hazards inherent in certain other physical modalities, and the supplying of large quantities (2–4 liters) of sodium chloride solution (0.6 per cent) by mouth during the treatment to replace chloride loss[6,11] have converted this form of therapy from one requiring hospitalization to one in which the patient is usually able to return to his work on the day following treatment, unless the extent of his disability makes hospitalization desirable. We have found that hospitalization for approximately two weeks is a distinct advantage in the management of acute gonorrheal arthritis. The need for hospitalization is usually not so urgent in cases of chronic gonorrheal arthritis.

THERMOLABILITY OF THE GONOCOCCUS

It has long been known that *Neisseria gonorrheae* is a particularly thermolabile organism. There are many records of spontaneous recovery or remission from the manifestations of gonococcal infections during intercurrent febrile disease. It is also well known that the gonococcus soon disappears and is replaced by secondary invaders when it reaches deep tissues, such as the fallopian tubes. Neisser and Scholtz[12] found it difficult to cultivate the gonococcus in patients with fever. The organism does not grow well on artificial mediums at temperatures above 100.4°F. (38°C.).

The recognition that fever exerts a favorable influence upon gonorrhea and its complications has led to treatment with fever produced by malaria inoculation or with chemical or foreign protein substances. While some satisfactory clinical results have been obtained by such methods, they have the drawback of being inconstant in their fever-producing properties and frequently provoke serious, uncontrollable, and occasionally fatal, reactions. Localized fever treatments have been carried out, for the most part with high-frequency electrical currents such as diathermy or radiothermy. The chief objection to this procedure is that the heat treatment is limited to a localized area. Patients with gonorrheal arthritis are suffering from a systemic disease, requiring systemic treatment.

The indefinite, and often contradictory, reports of the thermal death time of *Neisseria gonorrheae* have been clarified by the extensive thermal death time gradient studies made recently by Carpenter, Boak, Mucci and Warren.[13] When it became apparent that gonococcal infections responded to artificial fever therapy when the fever was sustained for long periods at a high level, these workers set out to determine the thermal death time gradient of the gonococcus when subjected to temperatures that can be tolerated by man. The *in vitro* thermal death time of 15 strains of *Neisseria gonorrheae* was determined at different

temperature levels. Some of the strains had been under artificial cultivation for many years, while others had been isolated one to four months previously. At 102.2°F. (39°C.) growth was not appreciably affected. At 104°F. (40°C.), about 99.7 per cent of the organisms were killed by ten hours' exposure. At 105.8°F. (41°C.), 99 per cent of the gonococci were destroyed in from four to five hours' exposure; the remaining 1 per cent required eleven to twenty-three hours. At 106.7°F. (41.5°C.) and 107.6°F. (42°C.), 99 per cent of the gonococci were rendered non-viable in two hours, while the remaining 1 per cent required five to twenty hours. The cultures which had been under cultivation for only a short period were most susceptible to heat. From these studies it became apparent that it would be possible, in most instances, to exceed the thermal death time of the organism without injury to the human host, by adapting the temperature level in patients with gonococcal infections to the thermal death time of the organism. We have repeated the experiments of Carpenter, Boak, Mucci and Warren with essentially identical results.

METHOD OF TREATMENT

During the past four years we have subjected 383 patients to 2844 artificial fever treatments (approximately 16,000 hours of sustained fever). With the exception of occasional mild skin burns, which occurred particularly at the beginning of this undertaking, no person has been injured by the treatments. Of these, 65 patients suffering from simple gonococcal urethritis or from gonococcal urethritis complicated by cervicitis, salpingitis, ophthalmia or arthritis, have been treated. This report is concerned only with the 31 patients who had gonorrheal arthritis. The favorable results obtained in the other manifestations of gonococcal infection will be made the subject of a subsequent report. Suffice it to say that the results are practically identical with those obtained by Desjardins, Stuhler and Popp,[14] of the Mayo Clinic, which moved Stuhler[15] to state in a later report: "I believe that the introduction of fever therapy for gonococcal infections is one of the greatest advances made in the last fifty years. I believe it is of even greater importance to the clinician than was the discovery of the gonococcus by Neisser in 1879."

While it would seem logical to adapt the treatment of each patient to the thermal death time of the strain of gonococcus producing his disease, there are important objections to such a plan. It requires several days to carry out accurate thermal death time determinations. Occasionally the organism is recovered with difficulty. Individual patients may harbor several strains, which differ in their heat-resistance. In rare instances, highly resistant strains require twenty or more hours at temperatures of 107°F. (41.7°C.) or above before they are destroyed. Moreover, it is quite likely that factors other than direct destruction of the gonococcus, such as stimulus to the defense mechanisms of the body, are responsible in part for the results achieved by fever therapy.

For practical purposes, we have found that four or five treatments, each of five to seven hours' duration, at a temperature range of 106°F. (41°C.) to 106.8°F. (41.5°C.), at intervals of three to five days, are productive of prompt and satisfactory results. Early in the course of this undertaking, it was our practice to give the treatments at intervals of one week or longer. The average number of treatments given to the patients in this series was approximately 5 (4.8). The average interval between treatments was approximately seven (7.4) days. Experience has taught us that fewer treatments are required if the interval between treatments is shortened. Four treatments at intervals of three to five days, the interval depending upon the general condition of the patient and his tolerance of the treatments, are usually regarded as a minimal course. If all evidence of activity in either the joints or the urethra

has not disappeared, additional treatments are given.

CLINICOPATHOLOGIC OBSERVATIONS

Included in the series which forms the subject of this report were 26 men and 5 women. The ages ranged from sixteen to forty-five years; the average was twenty-six years. Nineteen were suffering from the acute form of the disease, while 12 had chronic gonorrheal arthritis. The patients were not selected. The only requirements were that a diagnosis of gonorrheal arthritis could be established and that the physical condition of the patient would permit him to tolerate the treatments.

All patients are subjected to a thorough diagnostic survey to determine eligibility for fever therapy. Special studies are made of cardiac, vascular and renal functions, including electrocardiographic, basal blood pressure and blood chemical analyses.

Contrary to common belief, multiple joints were usually affected. In 23 of the patients (74 per cent) the arthritis was polyarticular. In the 8 patients with monarticular arthritis the knee was involved in 4 instances; the ankle in 2; the wrist in 1; and the foot in 1. In the polyarticular group one ankle was involved in 11 instances; both ankles in 5; one knee in 7; both knees in 2; one foot in 4; both feet in 4; one hand in 2; both hands in 6; one hip in 2; both hips in 5; one wrist in 5; both wrists in 2; one shoulder in 4; both shoulders in 2; one elbow in 3; cervical spine in 2; and sternum in 1. Even though there was no great difference in the ultimate response of the different joints to fever therapy, the weight-bearing joints were often much slower in returning to normal function.

During the routine preliminary diagnostic survey, diagnostic aspiration of the affected joints was done to determine the presence or absence of fluid. Pyarthrosis was found to exist in one or more of the affected joints of 8 patients. Gonococci were recovered by culture of the aspirated exudate from the knee joints of 2 patients with monarticular arthritis. An effort was made to determine the total white cell count and differential count on all aspirated specimens. Due to the abundance of fibrin in the aspirated fluid of a few of the patients accurate determinations could not always be made. The total leucocyte count and polymorphonuclear neutrophile count was much higher in the synovial fluid infected with the gonococcus. There was no appreciable difference in the total protein content in infected and non-infected joint fluids.

Twenty-one of the 31 patients had received some form of local chemical treatment of the genitourinary tract before the onset of arthritis. The treatment consisted of the instillation of some silver preparation into the urethra. Some of the patients with chronic urinary tract infection had received prostatic massage and treatment with sounds. A few of the patients attributed the onset of the arthritis to local traumatism or to some unusual exertion, such as lifting heavy objects. There was no evidence that these factors were of any importance in the onset of the disease.

In all of the 31 cases a definite history of previous gonococcal infection of the genitourinary tract could be elicited. At the time of the diagnostic survey prior to fever therapy the smears of 23 patients revealed the presence of gram-negative intracellular diplococci. Of the 8 patients with negative smears, 3 had acute gonorrheal arthritis and 5 had chronic arthritis. Of the 3 patients with acute arthritis, but negative smears, there were 2 men with acute prostatitis and one woman with acute cervicitis; all were reported to have had positive smears previous to their admission to the Fever Therapy Research Department. Of the 5 cases of chronic arthritis, with negative smears, all had had positive smears while under the observation of their physicians. In no case was the infection restricted to the anterior urethra in the male. Of the 5 female patients, there were 2 with pelvic cellulitis, 2 with acute cervicitis, and one with urethritis complicated by Bartholinian

abscess. Following the course of fever therapy the smears from the genitourinary tract of 24 patients showed no gonococci. The smears of 7 patients were still positive. Of these 7 patients, the smears of 4 became negative within 2 weeks without further treatment; 3 patients received local chemical treatment following the fever treatments. Two of the 3 patients receiving additional local treatment had received inadequate fever therapy; the third had received what was regarded as sufficient fever therapy, but the urethral smears still remained positive. This patient had severe folliculitis and posterior urethral stricture. After prostatic massages and the introduction of sounds, the smears became negative.

In all of the male patients prostatic massage was carried out at weekly intervals until pus cells and shreds did not appear in the urine. The average number of prostatic massages was 4. Complete genitourinary examinations were made on all patients at weekly intervals, including complete urine analysis and the two-glass urine test. Urethral sounds were passed and endoscopic examination was done in all cases before dismissal. Successive reexaminations have revealed no recurrence of gonorrhea in any of the patients.

The erythrocyte sedimentation time was determined routinely before fever therapy was instituted, at the completion of the course of fever therapy, and usually at intervals of two weeks until it returned to approximately the normal level. The Linsenmeyer method was employed, in which sedimentation time of three hours or more is regarded as normal. In the case of acute gonorrheal arthritis the average sedimentation time before fever therapy was twenty-one minutes; in the chronic cases, one hour and seventeen minutes. At the completion of the course of fever therapy the average sedimentation time for the acute cases was one hour and eleven minutes; in the chronic cases, one hour and forty minutes. The average interval for the return of the sedimentation time to normal was one month and nine days in the cases with acute gonorrheal arthritis. In the chronic cases, the average interval for the return of the sedimentation time to the normal level was one month and fourteen days. It will be observed that there is no essential difference in the time required for the sedimentation time to return to the normal level in patients with either acute or chronic gonorrheal arthritis. The sedimentation time of untreated patients with acute gonorrheal arthritis is distinctly shorter than in patients with the chronic form of the disease. While the sedimentation time is of no value as a diagnostic procedure, it provides a reliable guide to the response to therapy.

The average interval between the onset of gonorrhea and the onset of arthritis was one month and fourteen days in the acute cases, and two months and two days in the chronic cases. In one exceptional case there was a three-year interval between the initial infection and the first onset of the arthritis; this patient had experienced three recurrent attacks of gonorrheal arthritis and four recurrent attacks of gonorrhea, the last of which recurred four and one-half months prior to the institution of fever therapy.

The average interval between the onset of arthritis and the institution of fever therapy was eleven days in the acute cases, and two years and three months in the chronic cases. In 9 of the 12 chronic cases the average interval was four months and nine days; the other 3 patients had had previous attacks of gonorrheal arthritis extending over periods from four to thirteen years.

Of the 19 patients with acute gonorrheal arthritis, the average improvement in joint function immediately after the conclusion of the course of fever therapy was 77.6 per cent; in 3 patients the restoration of joint function was complete. Of the 12 patients with chronic gonorrheal arthritis, the average improvement in joint function at the conclusion of the course of fever therapy was 62.5 per cent; in 4 patients joint function was completely re-

stored. At the present time, the average improvement in joint function in the acute cases is 98.4 per cent; 13 patients have obtained complete restoration of joint function. In the chronic cases, the average improvement at the present time is 88.3 per cent; one additional patient has obtained complete restoration of joint function.

Worthy of particular mention are our experiences with 2 cases of chronic gonorrheal arthritis, in which almost complete limitation of motion of one knee joint remained after the usual course of fever therapy. After all evidence of active inflammation had disappeared the joints were manipulated under general anesthesia (brisement forcé) in order to separate the fibrous adhesions. Fever therapy was immediately instituted following the surgical manipulation. In both cases practically complete joint function has been restored. Orthopedic management is a requirement in those cases of chronic gonorrheal arthritis in which partial ankylosis has occurred prior to the institution of fever therapy.

SUMMARY AND CONCLUSIONS

1. High, sustained, controlled artificial fever is the treatment of choice for gonorrheal arthritis.

2. Gonorrheal arthritis is a manifestation of a systemic disease, requiring systemic treatment. *In vitro* thermal death time studies, and the clinical response of patients with gonococcal infections to artificial fever therapy, indicate that it is possible, in most instances, to destroy gonococci in the various lesions of the disease with high, sustained body temperature. In addition to this sterilizing effect, there is evidence that artificial fever therapy stimulates immune reactions.

3. Thirty-one patients with gonorrheal arthritis, associated with gonococcal infection of the genitourinary tract, have been treated by us with artificial fever therapy, utilizing a simplified, relatively safe and controllable air-conditioned apparatus, known as the Kettering hypertherm. Of 19 patients with acute gonorrheal arthritis the average improvement in joint function immediately after the conclusion of the course of fever therapy was 77.6 per cent; in 3 patients the restoration of joint function was complete. The ultimate average improvement in joint function in the cases of acute gonorrheal arthritis was 98.4 per cent; 13 patients have obtained complete restoration of joint function. Of the 12 patients with chronic gonorrheal arthritis the average improvement in joint function at the conclusion of the course of fever therapy was 62.5 per cent; in 4 patients joint function was completely restored. The ultimate improvement in joint function in the cases of chronic gonorrheal arthritis was 88.3 per cent. At the conclusion of the course of fever therapy gonococci had disappeared from the smears of the genitourinary tract of 24 patients. The smears of 4 patients became negative within two weeks after the last fever treatment. Supplemental treatment eliminated all evidence of gonococcal infection of the genitourinary tract of the remaining 3 patients.

4. In 2 cases of chronic gonorrheal arthritis almost complete limitation of motion of one knee joint remained after the conclusion of the course of artificial fever therapy. Orthopedic manipulation (brisement forcé) under general anesthesia was done to separate the fibrous adhesions. Artificial fever therapy was instituted immediately following the surgical manipulation. Practically normal joint function has been restored in both cases.

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[For Remainder of References see page 452.]

BACTERIOPHAGE THERAPY

A CLINICAL STUDY, WITH SPECIAL REFERENCE TO THE TECHNIQUE OF APPLICATION*

RALPH LAMPERT, M.D.

Instructor in Surgery, Louisiana State University School of Medicine

AND

FREDERICK FITZHERBERT BOYCE, M.D.

Assistant Professor of Surgery, Louisiana State University School of Medicine

WITH ELIZABETH M. MCFETRIDGE, M.A.

Secretary of the Department of Surgery and Instructor in Medical Literature, Louisiana State University School of Medicine

NEW ORLEANS, LA.

IN view of the prevailing attitude toward bacteriophage, which ranges from kindly skepticism to frank disbelief, one feels distinctly apologetic in presenting a purely clinical report on its therapeutic results. Even among those who are most open-minded the feeling is that the clinical application of the method is unwarranted until it is clearly understood why it does what it does—if it does anything at all—and until complete laboratory data are available for every case in which it is used. With those premises, it must be admitted, there can be no possible quarrel from the theoretic point of view. They are eminently scientific and quite in accord with the principles upon which the scientific practice of medicine should be based.

From the practical point of view, however, the situation is not quite the same. For one thing, bacteriophage would not be the first or indeed the only therapeutic agent whose effect had been utilized before its *modus operandi* had been demonstrated. For another, however unscientific the reasoning may be, the real worth of any system of therapy is estimated first, last and always by the clinical results it achieves. They are the patient's chief and only concern. What he is after, as Howard Kelly shrewdly puts it, is a cure, with or without a sound scientific theory behind it.

Finally, if full laboratory data must be secured in every case in which bacteriophage is indicated, delays in the institution of the treatment will be inevitable, and, as we shall point out later in this discussion, the full effectiveness of the method will be diminished. In the most competent hands laboratory studies take time. A culture cannot be made and bacteriophage cannot be matched against the demonstrated strain of bacteria without the loss of at least twenty-four hours and very often more, quite aside from the fact that the equipment and personnel necessary for such studies are not always available. We seriously question whether such delays are always worth what they cost; indeed, we believe that in some cases, at least, they cost more than they are worth. Surely in certain diseases certain assumptions as to bacteriology may be made with a high degree of accuracy, on the basis of the cumulative experience of the past, and we ourselves, acting upon these assumptions and instituting bacteriophage therapy with-

* From the Department of Surgery, Louisiana State University School of Medicine.

out delay, have many times aborted a lesion or materially shortened its course. Either event is clearly to the best interests of the patient, and the facile retort that infections which respond so promptly to such treatment would never have been serious ones seems to us neither fair nor valid. Undoubtedly some of them would not have been, but undoubtedly others would have been, and in such hypothetical circumstances one man's guess is at least as good as another's.

Recent communications dealing with bacteriophage in all its aspects make it entirely unnecessary to enter at this time into any detailed discussion of the various theories concerning its nature and its possible mode of action. The point which we desire to stress particularly in this report, that the manner in which bacteriophage is applied has much to do with the success or failure of the method, is also stressed in two of these communications, the series of articles by Eaton and Bayne-Jones[2] and the combined laboratory and clinical study by Jern, Howes and Meleney.[3] Eaton and Bayne-Jones, after commenting that too many successes have been reported with bacteriophage to make it possible to say it has no effect, remark that the variations in the reported results may be due to differences in the mode of application and in the dosage used. Jern and her co-authors say that success depends upon a combination of conditions intrinsic in the patient, in the bacterium and in the lytic principle available, as well as upon the manner in which these are brought together, and they set down as the minimum essentials for success a potent bacteriophage, a susceptible infecting organism, an adequate and intimate contact of bacteriophage with the infecting organism, and an environment favorable to or at least not antagonistic to the interaction of the two. Both groups of authors, in other words, emphasize, exactly as we do, that the manner in which bacteriophage is applied has much to do with the results which it achieves. Our own convictions on this point have been strengthened by our observation of the cases handled by bacteriophage on other services, in which the application was haphazard and, by our standards, entirely inadequate, and in which the results were almost uniformly unsatisfactory.

During the last two years we have used bacteriophage therapy in almost a thousand cases of superficial and deep infections, including carbuncles, furunculosis, infected wounds, breast, ischiorectal, perirectal and other abscesses, certain hand infections, and a miscellaneous group of other infections. In the miscellaneous group of cases, in which the application of bacteriophage was more or less tentative, the results were very uneven. In the other groups mentioned, however, the results were more than 90 per cent successful from the standpoint of promptness of relief, promptness of cure, and absence of recurrence. Except for the demonstration of the causative organism in enough cases to prove our contention that in certain diseases certain organisms can safely be assumed to be responsible, our use of this method has been entirely clinical, chiefly because, when we began, we could not secure the necessary laboratory assistance to permit detailed studies of every case. We have, therefore, no bacteriologic support for our clinical observations, but at that, certain data which we secured by the trial and error method are amply supported by the combined laboratory and clinical data which Jern and her associates report from their detailed study of 110 cases of carbuncles and furunculosis.

These authors found, for instance, that no difference exists between hemolytic and nonhemolytic strains of bacteria (staphylococci) in their susceptibility to bacteriophage. We worked on the assumption, confirmed in a small series of cases, that practically all carbuncles and furunculosis, as well as most hand infections, are caused by the staphylococcus, just as in most abscesses about the rectum the colon bacillus is the predominant organism. In the first group of cases, therefore, we em-

ployed a bacteriophage (polyvalent) potent against the staphylococcus, and in the second group we employed a bacteriophage (polyvalent) potent against the colon bacillus. It was soon evident that the application of a certain type of bacteriophage in a certain type of infection would usually bring about a certain highly desirable result, and increasing experience has served to confirm us in this assumption.

Jern and her associates, again, found that while bacteriophage may continue present in the lesion at the end of twenty-four hours, its potency is definitely diminished and it does not renew itself indefinitely but must be renewed. We promptly discovered that daily applications of bacteriophage in generous amounts were necessary for success, and that good results could not be expected unless the bacteriophage was brought into direct contact with the diseased tissues. The decrease in the potency of the bacteriophage, which is evidenced clinically either by failure to improve or by actual regression of the process, may be due to some bacteriologic cause as yet undetermined. It may perhaps be due to the inhibitory action of pus or serum, since liquefaction is the first result of bacteriophagy, and laboratory proof already exists that such an environment is hostile to bacteriophage. It may be, as we ourselves are inclined to believe, that the bacteriophage applied to the lesion is actually carried off in the discharge as liquefaction occurs. Whatever the reason, daily applications are necessary, and we arrived by the clinical route at precisely the same conclusions which Jern and her associates reached by their laboratory studies.

An illustrative case might be quoted in support of this contention.

A negro woman with a large carbuncle on the back was referred to us by a frankly skeptical member of our own staff. She was treated, by the usual routine, with daily applications of staphylojel and injections of staphylolysate and she showed such marked improvement that at the end of the third day she was turned over to the interne on the service, who was given detailed instructions for her further treatment. We were correspondingly surprised, in view of the excellent progress she had made under our observation, to be informed a few days later by the referring surgeon that the results of bacteriophage therapy in this case were just as unsatisfactory as he had expected them to be, and that he was scheduling the woman for surgical incision the next day. We were no longer surprised, however, when we found, on investigation, that the interne, considering our routine unnecessary, had taken it upon himself to withhold the daily injections and had used the bacteriophage only once since we had withdrawn from the case. When the proper routine was again instituted, the patient's progress to recovery was rapid and satisfactory, without operation.

Finally, Jern and her associates found that in most cases after the application of bacteriophage the characteristics of the invading bacillus are changed in the direction of lesser virulence, but that in a small percentage of cases the organism may become resistant to the lytic action of the bacteriophage. In a certain percentage of our own cases the results were not satisfactory and we have come to believe that in these conditions bacteriophage is not indicated and should not be used. In another group of cases, chiefly osteomyelitis, our results have been uneven, though usually some improvement has been evident, which we interpret as due to "the lessened virulence" of the invading organisms. In most conditions, however, in which we have learned by experience to anticipate good results, we have come to believe that unsatisfactory results are more often due to the improper application of the bacteriophage than to the development of any resistance by the organism, and it is for that reason we desire in this communication to outline the method of application which has in our hands given the best results.

We plead guilty at the outset to the charge of using commercial preparations. We see no more reason, to speak frankly, for not using bacteriophage made by

reputable pharmaceutical and biological firms than for not using sera and vaccines and catgut prepared by them. It is quite obvious that unless commercial preparations can be used, bacteriophage therapy must remain a form of treatment limited to physicians with access to laboratories and laboratory assistance, advantages which the majority of medical practitioners do not possess. The use of a polyvalent phage, containing one predominant strain in combination with less dominant strains, takes care of the objection that the wrong phage is being used. It is our impression that the liquid bacteriophage (lysate) is rather more effective than the jelly and we use it whenever practical, but the jelly is more satisfactory in the treatment of open wounds than is the old method of moist dressings or the instillation of fluid phage into open cavities in which it cannot be expected to remain. It might be added, merely for the sake of argument, that if, as some authorities contend, bacteriophagy is simply another form of foreign protein therapy, the bacteriologic aspect becomes less important and the whole question of commercial preparations seems much ado about very little.

In the endeavor to bring the bacteriophage into direct contact with the lesion we have used all possible methods, including external application, direct injection, and circuminjection, and always, in favorable cases, with the same results. Within the first twenty-four hours of the application, which is made as soon as the patient is seen and regardless of whether fluctuation is evident or the stage of induration is still present, marked liquefaction occurs, and necrotic material can be aspirated or expressed in varying quantities at the time of the second treatment. As the treatment is continued the spread of the infection is arrested, induration steadily decreases, sloughs liquefy and separate, leaving clean, granulating surfaces, and, except in very extensive lesions, the patient is usually clinically well within a week.

We grant, as Jern and her co-authors point out, that the diseases for which we advocate bacteriophage therapy are self-limited diseases and that in them three stages are usually noted, induration, liquefaction and discharge, and resolution. We agree with them also that merely because this course occurs under bacteriophage therapy it does not follow that the therapeutic agent has brought it about. On the other hand, even in self-limited diseases the progress to recovery can be hastened by various therapeutic measures, and it should be so hastened, for many times such diseases, if they are left to natural processes, may cause considerable damage. A carbuncle, for instance, can kill a diabetic, and even in a normal individual it can cause long-continued disability, associated with a deforming loss of skin and with more or less pain and morbidity.

The amount of pain which carbuncles, abscesses and similar lesions can cause to the patient should not be taken as lightly by the physician as it frequently is. We stress that point, for in our experience one of the most striking results of bacteriophage, undoubtedly the result for which the patient is most grateful, is the very prompt relief of pain which follows its use. This feature, for some reason, is not mentioned in any of the papers on the subject which have come to our attention, yet in none of our successful cases did the pain of the lesion amount to anything more than discomfort after the second or third day of treatment, while in many instances relief was reported within a few hours, and in a small group of cases, which we mention with hesitancy because they sound so fantastic, relief was almost immediate.

This contention will be best proved by one or two illustrations.

One of them concerns a medical student, whose intelligence is well above the average. When he was first seen he had a large carbuncle on the back of his neck, he was unable to move his head, and he looked haggard and ill from pain and loss of sleep. He was treated by our usual technique and returned the following morning looking refreshed and well; he was able to move his head freely in any direction and he asserted that he had slept the clock

around. Liquefaction was marked, the dressing being saturated, and a considerable quantity of pus was expressed before further staphylolysate was injected. Sometime later, in discussing bacteriophage therapy in his presence, we mentioned that in his case we considered that cure had been complete within a week. From his standpoint, he said, it had been complete within four hours, for after that time he had been practically free from pain.

Christopher sets down in italics as one of the diagnostic signs of bone felon pain in the finger tip *so severe as to interfere with sleep.*

A patient who exhibited that feature of the lesion presented himself at the clinic one morning, so completely unnerved from several days and nights of torturing pain that the first bacteriophage treatment was done with the greatest difficulty. He was instructed to return to the department office next morning at ten o'clock for the second treatment. He arrived, instead, at three o'clock in the afternoon, full of apologies, and with the explanation that he had slept the night through, and part of the day as well, and that his family, knowing what he had previously endured, had refrained from waking him. His appearance and his altered nervous equilibrium bore witness to the truth of his story, as did the fact that his finger could be manipulated as we wished. Cure, without deformity or scarring, was complete in ten days.

Such instances, regardless of how incredible they sound, we have noted again and again, and we set down as the most immediate clinical result of bacteriophage therapy a relief of pain which has occurred too promptly and in too many cases to be attributed to imagination or psychic effect or anything other than some definite analgesic action in this agent.

We might say that when bacteriophage is injected the patient practically always complains of pain and burning, which is sometimes quite severe. It is transitory, however, usually disappearing completely by the time the dressing is completed, and we do not consider that it warrants the additional discomfort which would be associated with the use of a local analgesic agent for its relief.

We have no control series in the sense that we can present for every case of every lesion treated by bacteriophage therapy a similar case treated by some other method and alike in extent, stage of progress and virulence of the offending organism. Only a control series of that sort would furnish adequate grounds for comparison, and the difficulty, if not the impossibility, of securing it need scarcely be pointed out. We have, however, selected certain groups of cases treated by bacteriophage and have compared them from the point of view of duration of treatment with similar groups of infections treated over by the same period of time by surgical measures. It should be remembered that all the patients in the control group were hospitalized, whereas more than half of the patients treated by bacteriophage were ambulatory, although under the usual methods of treatment most of them would have been hospitalized. Practically all the patients in the surgical group, furthermore, had some sort of anesthetic, usually ethylene or ether, whereas none of the patients in the bacteriophage group had any sort of anesthesia or analgesia. Aside from any other consideration, therefore, the advantage clearly lies with the bacteriophage group from the standpoint of economy and convenience, and we believe that in extent and severity of the infectious process the two groups of cases are practically even.

In the cases of breast abscess treated by bacteriophage the period of treatment (treatment in all cases meaning the entire period the patient was under observation) averaged six days, against an average hospitalization of 12.5 days for the cases treated by surgical measures. In the cases of ischiorectal and perirectal abscess treated by bacteriophage the period of treatment averaged five days, against an average hospitalization of thirteen days for the surgical cases. In the carbuncles treated by bacteriophage (they varied from 2 to 12 inches in diameter) the period of treatment averaged eight days, against an average hospitalization of twenty-seven days for the cases treated by surgery, in a

few cases with the addition of vaccine therapy. In almost all the cases treated by surgical measures the patients were discharged to the clinic for further observation and treatment, whereas all the patients treated by bacteriophage were finally discharged, with instructions to return only if there was a recurrence of the infection. The percentage of recurrence, we might say, in the cases treated by bacteriophage was under 2 per cent.

It is only fair to add that there were two deaths in the group of carbuncles treated by bacteriophage, against three in the surgical group.

One patient was a white male, fifty-seven years old, who had a severe diabetes and who when he was first seen exhibited a carbuncle which involved the whole back of the neck and extended up into the scalp. It responded promptly to bacteriophage therapy, and at the end of three weeks, when the entire involved area was represented only by clean, granulating tissue, he was transferred to the medical service, on which he died a month later from an acute nephritis superimposed upon his diabetes.

The second death occurred in a negro female, also a diabetic, and was due to septicemia; in this case surgical incision was used as well as bacteriophage.

The technique of application varies according to the location of the lesion, but the aim, as we have said, is always to bring the bacteriophage into intimate contact with the lesion, and daily applications are always made during the first days of treatment. In bone felons, tenosynovitis, collar button abscesses, acute paronychia and other hand infections in which it seems possible to bring the bacteriophage into adequate contact with the infected tissues, a small incision is made over the involved area, such pus as may be present is expressed by very gentle pressure, and the wound is filled with staphylojel, the treatment being repeated daily as long as the necessity for it is evident.

We have had excellent results with this method, particularly in bone felons, in which the classical incision always causes a deforming scar and more or less permanent functional disability. In all the cases we have seen early, and in several rather advanced cases, the functional result has been practically perfect, though naturally the later the case is seen the less good are the results. We would emphasize, however, that if the infection has advanced to such a point that it is doubtful whether the bacteriophage can be brought into contact with all the involved tissues, the method is useless, and should not be attempted. This was clearly proved to us by one of our first patients, a negro woman who finally required amputation at the wrist.

She sought treatment three weeks after the original injury, and she had been subjected to two extensive surgical incisions when we first saw her. The infection involved practically the whole hand, and although some improvement was evident after the use of bacteriophage, the results were unsatisfactory, and a third incision and finally amputation were required.

In such a case we would realize now, as we did not then, that we were foredoomed to failure, for it was clearly impossible to introduce the bacteriophage so that it would reach all the infected tissues. Another hand infection resulted fatally.

The patient, a negro female, died in a diabetic coma after two incisions for drainage, the first before admission to the ward and before the application of bacteriophage, which, as in the case just mentioned, was merely an auxiliary measure.

In furunculosis local applications of staphylojel are used if the lesions are large enough to warrant local treatment, and in the axillary variety, which is notoriously resistant to treatment, direct local injection of staphylolysate is also used. In this type of case, however, our chief reliance is on intramuscular injections of the staphylolysate. The initial dose of 0.5 c.c. is increased to 1 c.c. the second day and by 1 c.c. each successive day until a maximum of 5 c.c. is attained. This dose is given for at least five days, and in particularly obstinate cases for three or four days longer. Some of the patients in this group had had recurrent infections for more than

a year, and the outstanding case of the series was a pharmacist who had been annoyed by repeated outbreaks for more than five years and who had submitted to every conceivable form of treatment without result. He was relieved by a single course of injections and has had no recurrence in the fifteen months which have elapsed since his treatment.

In all abscesses, no matter what the location, the technique is the same. Lysate (cololysate for abscesses about the rectum, staphylolysate for most other types) is injected into the area of induration, the same procedure being followed after the aspiration of the pus present if the stage of fluctuation has already been reached. Liquefaction is practically always evident on the second day, and the treatment thereafter consists of aspiration of the pus and injection of the lysate through the same needle, which is not removed from the abscess cavity during the maneuver. In some cases we have used as much as 20 c.c. of lysate at a single injection and in some ischiorectal abscesses in which no fluctuation was evident at the first treatment we have removed as much as 50 c.c. of purulent matter at the second visit. Rice makes the point, which we corroborate from our own experience, that the closed treatment of pus is perfectly safe; indeed, when it is possible, we believe that it is superior to the open method because secondary infections do not occur.

Infected sebaceous cysts and infected pilonidal cysts are treated as if they were abscesses.

In one case in the latter group the cyst wall was discharged as a slough about 2 inches in diameter. The odor was intolerably foul when the patient was first seen and the initial treatment was given with great difficulty because of her pain and discomfort; she left the hospital at the end of ten days with a perfectly clean wound.

We have been particularly impressed by the contrast between this case and two others recently treated on another division of our service by repeated incisions for drainage followed by radical excision of the cyst. One patient was in the hospital for five weeks and another for seven weeks, and both required treatment for several weeks longer in the clinic.

In carbuncles the procedure varies according to the size of the lesion. In those smaller than 2 inches in diameter staphylolysate, usually in quantities less than 1.5 or 2 c.c., is injected directly into the necrotic area, care being taken to prevent any increase in tension and to avoid the blood vessels in the involved area. Staphylojel is also applied locally. Usually on the second day a considerable quantity of pus can be expressed, which is replaced with 3 to 5 c.c. of lysate. The procedure is repeated daily until the liquefied slough is expressed as thin, purulent material through the multiple openings of the lesion.

In carbuncles varying from 2 to 12 inches in diameter, the latter being the largest we have treated, the initial treatment is the same as for smaller carbuncles, daily local applications of staphylojel and daily injections of staphylolysate following the expression of liquefied necrotic tissue. Usually by the third day a small incision can be made in the center of the carbuncle, and through it radial injections of staphylolysate are made through the infected tissues into the circumferential healthy area. The procedure, we grant, seems entirely illogical, if not actually dangerous, but in only one case has extension of the infection followed it, and that extension might, of course, have occurred even without the treatment. After three or four days of these radial injections, with continued local applications of the jelly, the slough is usually so softened and separated that it is possible to lift the skin with forceps and cut the slough away beneath it, the procedure being repeated daily until the entire slough can be removed. The importance of the radial injections and of the removal of the slough by this method can be proved by a comparison of the cases treated by this method with the early cases, in which it was not employed. In the early cases there was prompt relief of pain but the duration of treatment was twice as

long as in the later cases in which this technique was used routinely.

It should be emphasized that during the whole treatment the patient is increasingly comfortable, and that all the manipulations are carried out without anesthesia or analgesia. If the lesion is seen before loss of skin has occurred, the results are usually excellent and the skin loss is negligible, if any, the involved subcutaneous area being promptly filled in with healthy granulations. Of course, if skin has been lost before the patient is seen, this mode of therapy cannot replace it.

Of the miscellaneous group of cases in which bacteriophage gave uneven results we shall mention only osteomyelitis, of which we have treated 12 cases. In all of them surgical measures were used also and in all improvement was evident in that drainage decreased, the odor was less annoying and the patients were decidedly more comfortable, but in only 3 cases was the cure, if one dares to speak of a cure in osteomyelitis, apparently complete. Included in this group is one case of acute osteomyelitis which was treated with staphylojel after exposure of the bone and institution of drainage through a number of drill holes. The wound was healed and the patient apparently entirely well within three weeks, which is a remarkably short duration for osteomyelitis.

One final point should be mentioned, which is brought out by Jern and her associates and with which most authorities, we believe, are in accord, that the reaction to bacteriophage, while it may be favorable or indifferent, is never harmful. Bacteriophage therapy may not succeed but if it does not, it does no damage and it does not prevent the later use of other methods. It is scarcely justifiable to employ a therapeutic agent merely because it is innocuous, but it is another matter to employ it on the premise that it succeeds in most cases in which it is indicated and does no harm in those in which it fails. That has been our experience with bacteriophage. In the light of our own results we have come to believe that it is the method of choice in most cases of carbuncles, furunculosis, breast, ischiorectal, perirectal and miscellaneous abscesses, infected wounds, whether operative or traumatic, and hand infections in which it seems probable that it can be brought into direct contact with all the infected tissues. Unless it is used, however, according to the principles we have outlined and by the technique we have described we do not believe the results are likely to be uniformly satisfactory.

SUMMARY

1. Our clinical experience with bacteriophage in the treatment of certain types of infections, chiefly carbuncles, furunclosis, abscesses and certain hand infections, is based on approximately a thousand cases and has resulted in more than 90 per cent of successes.

2. We consider our successful results to be due in large measure to the technique of application which we employ routinely, and which is based upon direct contact of the bacteriophage with the infected tissues by means of generous daily applications.

3. The prompt relief of pain and the brief duration of treatment by bacteriophage therapy, as compared with the duration of treatment by other measures, are outstanding clinical facts.

4. The method does no harm in those cases in which it does not succeed.

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PLANTAR WARTS, CALLOSITIES AND CORNS

JOSEPH JORDAN ELLER, M.D.

Assistant Professor of Dermatology, New York Post-Graduate Medical School and Hospital, Columbia University

NEW YORK CITY

AMONG the common skin lesions of the feet are plantar warts, corns and callosities. While these conditions are not serious, they may occasion so much discomfort, and handicap the person's efficiency to such an extent that they are worthy of careful consideration.

PLANTAR WARTS

It is generally agreed that plantar warts and common warts are both due to an infection by a filterable virus. The plantar wart differs microscopically and clinically from the common wart only because it is found on the plantar surface of the feet where it is subject to constant pressure. Since they are infectious, the plantar and common warts are usually multiple. This is one of the points differentiating the plantar warts from the plantar callosities which usually appear as single lesions. On account of their infectious nature, it is not unusual to find the common warts on the hands and the plantar warts on the feet of the same patient.

TREATMENT

The treatment par excellence is destruction of the plantar wart by means of electrocoagulation. This can be done by anesthetizing the lesion with novocaine solution, following which the wart is destroyed by the electrical method. Qne treatment usually suffices, for recurrences with this method are rare.

The plantar wart may also be removed by the surgical scalpel. The disadvantage of surgical therapy is that it usually disables the patient for at least a week. Many patients are, therefore, averse to surgical procedures, and in these cases one may resort to another form of therapy which will not oblige the patient to keep off his feet. Among such methods the following may be mentioned: roentgen rays, radium, bichloracetic acid, trichloracetic acid, and salicylic acid plasters. In the literature on plantar warts claims are made for their cure by intramuscular injections of bismuth, and the internal administration of mercury. My experience with these methods has not corroborated such claims.

On very rare occasions plantar warts, like common warts, disappear spontaneously, and this fact has led to the use of various methods of "suggestion" therapy. I have never been able to corroborate the success of psychotherapy in the removal of common and plantar warts.

CALLOSITIES (CALLUS)

Callosities are hard, yellowish, circumscribed patches composed of horny masses. They usually occur on parts subjected to long-continued pressure, especially over bony prominences. A callus differs from a corn in that it has no central core, the overgrowth usually being diffuse.

Constant pressure over a bony prominence causes a simple hypertrophy of the corneous layer. The remaining layers are usually unaltered, or somewhat atrophic due to pressure.

Pathologically, the papillae are flattened, but seldom exhibit inflammatory changes. The squamous cell layer is thinned, and the stratum granulosum thickened. The corneous layer is enormously hypertrophied, with more or less accompanying condensation.

Callosities tend to disappear spontaneously when the cause is removed. To prevent recurrences this should be done before treatment is instituted. The prescribing of the correct shoes by an orthopedist is often necessary.

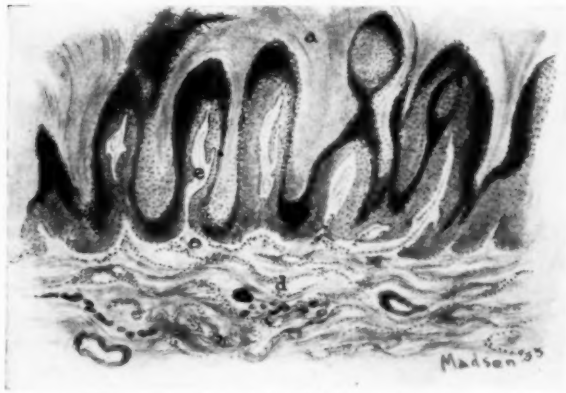

Fig. 1. Plantar wart. (a) Thickened corneous layer. (e) Elongated papillae. (d) Normal upper cutis.

The treatment consists in trimming the callus as thin as possible. This is followed by the application of 40 per cent salicylic acid plaster, which dissolves any new horny tissue which tends to form. The use of bichloracetic acid is sometimes valuable. This acid is an excellent keratin solvent, but must be used with care to avoid any contact with the normal skin. The acid is painted on the callus, the surrounding skin is covered with a protective layer of petrolatum, and the callus will shrink and harden. Some days later the lesion is shaved down with a sharp scalpel and the acid reapplied. This procedure is used at five day or weekly intervals. In stubborn and long-standing cases, irradiation in the form of radium or roentgen rays applied to closely shielded lesions often results in their absorption.

CORNS

Corns are in reality callosities in whose center cornification is highly developed to form horny, conical plugs. They arise at the site of constant pressure of the plug on the underlying nerve fibers. Primarily the growth is an attempt to protect the part from pressure. There is always the factor of constant pressure plus the presence of a bony prominence close to the skin in the causation of the growth of a corn.

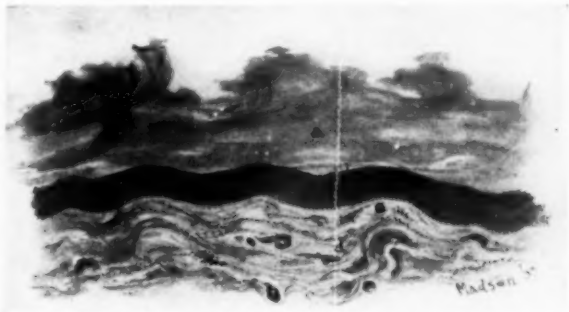

Fig. 2. Callus. (a) Marked hyperkeratosis. (b) Thickened granular layer. (e) Flattened papillae. (d) Corium with slight inflammatory changes.

Partially subluxated joints, bony spurs, and hypertrophic processes from arthritis are found frequently associated with corns. The ring of callus about corns and warts is secondary to the abnormal growth.

Structurally corns are composed of closely packed epidermal cells arranged concentrically. The horny layer of the epidermis is thickened and polished. The base of the corn is directed outward; the apex is fairly sharp and points inward. The presence of air in the layers of the corn usually gives it a white appearance.

At the base of the lesions the papillary bodies commonly are flattened, but surrounding the growth these dermal projections are enlarged, and their blood vessels dilated. Occasionally a coil gland outlet is found winding its way upward through the keratotic mass, but in the majority of instances these canals exist only in the peripheral regions. The glands themselves are well preserved, although both the coils and ducts are usually dilated. The connective tissue is condensed, and the elastic fibers have completely disappeared.

There are two varieties of corns: the hard and the soft. Hard corns are more common, usually occurring on the dorsal surfaces of the toes and the plantar aspect of the foot.

Soft corns are found between the toes. Histologically they are identical with hard

corns. Due to constant heat and moisture, they become grayish and macerated. Occasionally they are the seat of inflammatory reaction and are then known as "suppurating corns." Corns ache occasionally even at night while not under pressure.

FIG. 3. Corn. (a) Conical core of horn. (e) Loss of papillae from pressure beneath this area. (d) Slight inflammatory changes in corium.

Removal of the cause, and the wearing of corrected shoes is the first step in the treatment of corns. Any deformities of the foot should receive proper attention. This alone may cause a disappearance of the lesion.

There are numerous methods for the removal of corns. It may be done very successfully by the use of the scalpel or by the use of electrocoagulation. A more conservative method consists of softening the corn by frequent soaks in warm water, after which the area is pared with a knife until a punctate hemorrhage appears. This spot is then cauterized with trichloracetic acid. Occasionally this suffices for a cure. Corns may also be successfully removed by the application of a 40 per cent salicylic acid plaster placed over a period of several weeks.

Roentgen rays or radium may also be used, but I believe that the more conservative methods of therapy such as the aforementioned are preferable.

Carbon dioxide may be used in the treatment of corns as well as in the other conditions mentioned, but the percentage of successes with this form of therapy is poor as compared with other methods.

Suppurative corns, which are due to secondary infection, and are usually found between the toes, are best treated by prolonged immersion in warm antiseptic solutions such as 1:4000 bichloride of mercury, or 1:2000 potassium permanganate, following which the lesions are excised by the scalpel.

SUMMARY

1. A brief description of the differentiation, clinically and pathologically, between plantar warts, callosities, and corns has been given.

2. Various forms of therapy have been discussed, and rated according to their effectiveness, desirability, and the success to be expected.

FORENSIC MEDICINE

EDITOR: HARRISON S. MARTLAND, M.D.

INTRODUCTION BY THE EDITOR

THE REPORTING OF COMMON AND UNUSUAL MEDICOLEGAL CASES BY PHYSICIANS AND SURGEONS

FOR several years the writer has conducted, at the Newark City Hospital, weekly clinico-pathological conferences with the aid of the Attending and Intern Staffs and his associates in the hospital laboratories and Medical Examiner's Office of Essex County.

These meetings are open to any physician in the community or elsewhere who cares to attend. They start at nine o'clock in the evening so that doctors will not miss their evening office hours, and frequently last about three hours.

Interesting clinical cases from the wards and clinics illustrating almost every field of medicine and surgery are shown, their histories being read and their physical findings demonstrated by the interns. An open discussion follows calling for a differential diagnosis and the prognosis and treatment are briefly reviewed.

A brief report on follow-ups in cases shown at previous conferences is an important feature, the necropsy findings being shown whenever available.

The last half of the meeting is given over to the presentation of autopsy material from interesting cases occurring during the week. The clinical phases of the cases are evaluated and both gross and microscopic findings demonstrated.

In other words these meetings are conducted along lines similar to the well known CPC's now in use in almost all teaching institutions.

We feel that too often over-extensive detail in the presentation of a case makes the diagnosis so obvious that the listening physician finds his interest dulled and his keenness less sharp. If something is left to the imagination of the listener, he remains constantly in the picture and on the alert. A variety of cases at each clinic, in our opinion, makes it more certain that every doctor present will take home with him something of value to his daily work.

For years we have presented at each meeting the histories, photographs or actual specimens of medicolegal cases from the Medical Examiner's Office.

We have done this for several reasons: (1) To afford interesting variety with the consequent mental rest—incidental to the consideration of types of cases with which the average practitioner is unfamiliar but must occasionally meet. (2) To stimulate interest in forensic medicine, unfortunately a woefully neglected field in this country. (3) To demonstrate that important medical and surgical facts of constant value in the practice of medicine may be obtained from a Medical Examiner's Office.

The foregoing brief description of our work in Essex County and Newark, N. J. is the basis of a plea for greater cooperation between constituted hospital and medicolegal authorities in the reporting and publishing of cases of interest in forensic medicine.

It is unfortunate that because of this lack of cooperation a vast amount of material of deep scientific interest is lost. Medicolegal knowledge in this country is

still mediocre but is rapidly becoming one of the outstanding branches of modern science. Its importance in the detection of crime is poorly understood by laymen and physicians alike. With the material available in the United States an awakened interest by the medical profession would soon place this country in the forefront in these matters.

The cases reported in this issue are a few of the many presented from time to time before these conferences. We have asked those who are most familiar with the individual case to report them in narrative style.

For obvious reasons all identification marks such as name, initials of patients, dates, etc., have been omitted. The authenticity of any of the presented facts may be established at any time through our office.

The success of our department of forensic medicine depends primarily on an adequate supply of material for our pages. Any article dealing with a subject properly in the field of forensic medicine and of interest to surgeons will be welcome and will receive, if requested, the combined interest and efforts of our editorial staff in its preparation for publication.

Expression of approval or criticism in our conduct of this department will be gratefully received by the editors and publishers.

Harrison S. Martland

FOREIGN BODY IN RECTUM AND SIGMOID

DAVID A. KRAKER, M.D.

Attending Proctologist, Newark City and Beth Israel Hospitals

NEWARK, N. J.

THE specimen shown (Fig. 1) consists of a half pint whiskey bottle around the neck of which is tied 10 inches of heavy curtain cord having a loop at its other end. Hooked into the mouth of the bottle is a double strand of heavy galva-

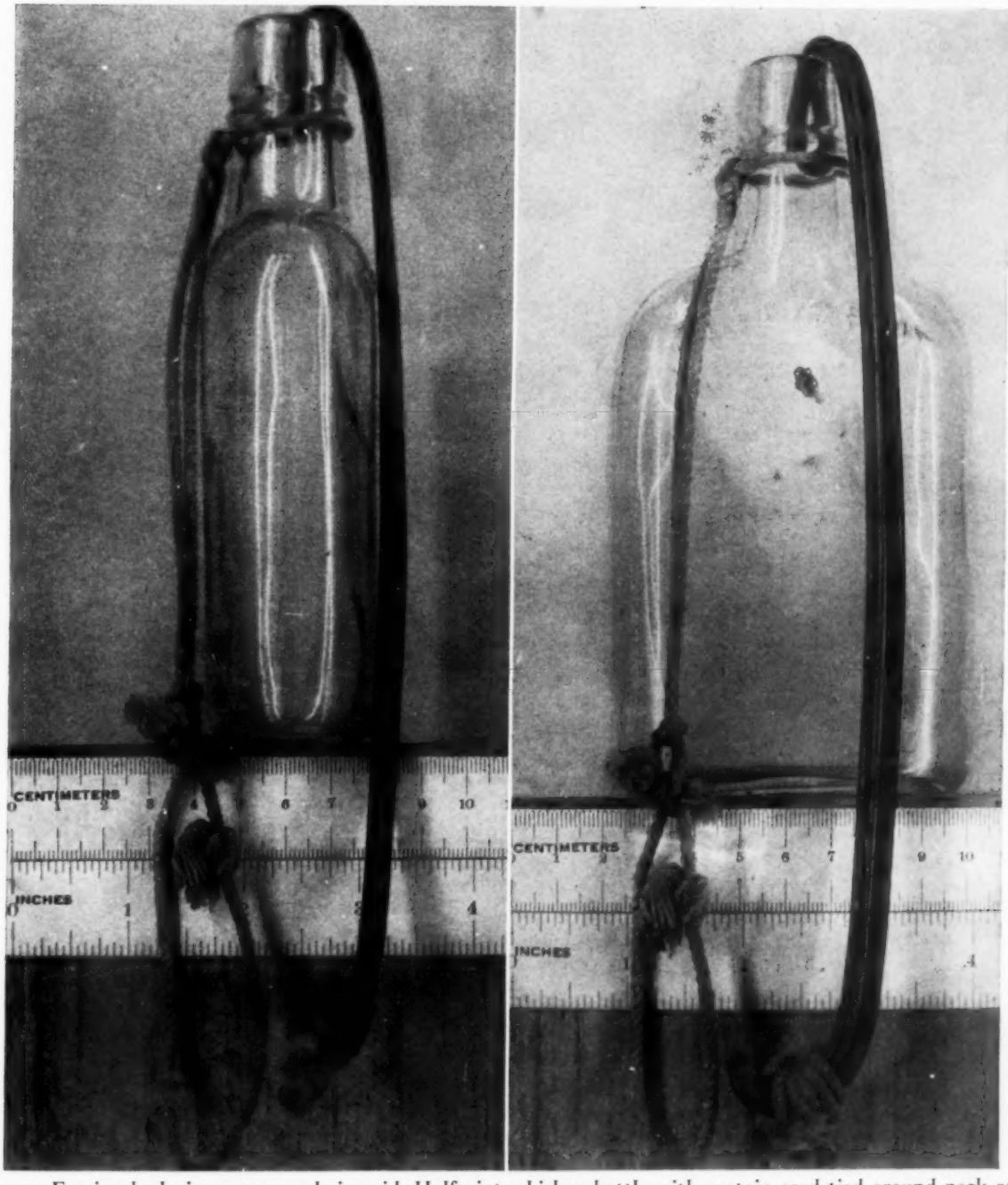

FIG. 1. Foreign body in rectum and sigmoid. Half pint whiskey bottle with curtain cord tied around neck and galvanized iron wire hooked into its mouth. Removed at operation from rectum and sigmoid of supposed sexual pervert. Photograph at left shows side view of bottle to illustrate relative positions of cord and wire, ends of which protruded from anus on admission to hospital. They were placed on bottle by patient to facilitate removal. (Office of Chief Medical Examiner, Essex Co. (Newark), N. J.)

nized iron wire, 3/16 inch in diameter and 10 inches in length. The bottle measures 6½ inches in length, 3 inches in its greatest width and 1½ inches in its greatest thickness.

Fig. 2. Same case. Small snapshot of patient made in ward on admission to hospital. Loop of curtain cord can be seen extruding from anus. On account of very poor photograph, end of iron wire which was also protruding cannot be seen. (Office of Chief Medical Examiner, Essex Co. (Newark), N. J.)

This bottle with the wire and cord attached was removed from the rectum and sigmoid of a white man fifty-one years old.

The patient was admitted to the Proctologic Service of the Newark City Hospital suffering from obstipation.

He stated that for ten days previous to admission he had had no bowel movement. Four days before admission, he attempted to relieve this condition by inserting a bottle into the rectum. In order not to lose control of the bottle he placed a wire hook into the mouth and tied a loop of curtain cord about its neck. His intention was to use both the wire and the cord as aids in removing the bottle. Repeated attempts to withdraw the bottle after its insertion failed. He had not defecated since its introduction and had vomited several times in the last few days. It should be noted that the first history obtained in this type of case must always be viewed with suspicion and doubt.

On admission he was not in any pain and had no chills or fever. Rectal examination showed considerable induration about the anal region. Protruding from the anus was a loop of curtain cord about 2 inches in length and the end of a double strand of galvanized iron wire (Fig. 2). On digital examination, about 3 inches above the internal sphincter, an obstruction was felt which seemed to be the bottom of a round glass bottle. Traction on both wire and cord, which were on opposite sides of the bottle, failed to move it from its fixed position. There was no evidence of bleeding or discharge.

The patient was taken to the operating room and under gas, oxygen, ether anesthesia, further attempts were made to remove the bottle by traction on the iron wire and cord. The bottom of the bottle was felt as an elliptical object about 1½ by 3 inches in size. It would not budge and was apparently held firmly in its position above the spine of the right ischium.

An incision was then made in the midline extending through the posterior rectal wall down to the coccyx. By means of traction upon the protruding wire and cord, aided by pressure from above, and the use of leverage the bottle was slipped over the spine of the right ischium. A half pint whiskey bottle, with curtain cord tied around its neck and the galvanized wire hooked into its mouth, was delivered "en masse." Bleeding points were tied and the wound was completely closed.

The patient made an uneventful recovery except for slight secondary hemorrhage occurring on the fifth day after operation. He was discharged, thirty-nine days after admission to hospital, cured.

Further questioning of the patient during his convalescence revealed a partial admission that this was not the first time a foreign body had been introduced by him, or by others with his consent. He gave a hazy account of companions who when drunk did such things for amusement. Although a definite admission was never obtainable it is assumed that this accident occurred as the result of sexual perversion.

From the surgical viewpoint the case is of interest because of the large size of the foreign body used; of the impinging of the base of the bottle above the ischial spines making removal difficult; of the high position of the bottle, the neck resting in the sigmoid; of the removal without breaking and the complete cure with no ill effects after forcible dilatation of the rectum and especially the rectosigmoidal junction.

FOREIGN BODY IN RECTUM

ARTHUR W. WYKER, M.D.

Attending Surgeon, St. Mary's (Orange), Mountainside (Glen Ridge), and St. Vincent's (Montclair) Hospitals

BLOOMFIELD, N. J.

THE specimen shown (Fig. 1) consists of a 3-ounce rectangular cold cream jar in the top of which is inserted an ordinary lemon. This was removed by sigmoidostomy from the rectum of a male, white, Greek, forty-eight years of age.

FIG. 1. Foreign body in rectum. Three ounce cold cream jar with lemon in its open end. Pushed up rectum by supposed sexual pervert, who stated he followed drug clerk's advice for cure of hemorrhoids by using cold cream and lemon juice. Removed through sigmoidostomy. (Office of Chief Medical Examiner, Essex Co. (Newark), N. J.)

The patient was admitted to the Surgical Service of St. Mary's Hospital, Orange, N. J., with a diagnosis of foreign body in rectum and intestinal obstruction.

He spoke very little English and complained only of pain in the rectum. Rectal examination showed the sphincters soft and dilated. A hard glassy object, feeling like a jar, was felt 3½ inches above the internal sphincter.

FIG. 2. Same case. Roentgenogram showing cold cream jar in rectum. Note that bottom of jar is directed downward and its top reaches pelvic brim. Faint outline of lemon marked by arrows is just visible. (Office of Chief Medical Examiner, Essex Co. (Newark), N. J.)

Roentgenographic examination showed a glass jar in rectum situated about 3½ inches above sphincter with its top pointing upward and reaching the pelvic brim. There was a suggestion of the outlines of an elliptical mass above it, which the patient stated was a lemon (Fig. 2).

Questioned as to how the foreign bodies got into his rectum, he simply stated that he put them there himself and refused to answer any other questions. We assumed they were placed there about twelve hours before admission to the hospital.

Several futile attempts were made to extract the jar by manual and instrumental manipulation through the rectum. The base of the jar was directed downward and covered with grease, causing all instruments to slip off.

The abdomen was distended but not hard. There was slight muscular rigidity over the symphysis. Leucocyte count 8920 per cu. mm.

with 68 per cent polymorphonuclear neutrophiles. Temperature 100.2°F., pulse 100 and respirations 18 per minute.

The patient was taken to the operating room and under ether anesthesia a median laparotomy was done. The position of the foreign bodies was ascertained and the sigmoid flexure brought into the wound. Abdominal pads were inserted and the adjacent intestines well walled off.

The sigmoid was then incised along its long axis and a large, swollen, juicy lemon 3 inches in its long diameter removed. A sponge forceps was then inserted through the opening in the sigmoid and passed into the rectum. The open top of a glass jar was seized and with the aid of an assistant pressing from below with his fingers in the rectum, a 3 ounce opal glass cold cream jar was removed through sigmoid. The patient left the operating room in good condition.

The first few days postoperative were a bit hectic. For three days he passed a small amount of bloody urine which cleared up.

Eight days after operation, his temperature was normal, and sixteen days after he was walking about the ward. He was discharged twenty-two days after admission, cured.

During convalescence he had time to think up a story. A few days before admission to the hospital he asked a drug clerk what to do for a bad case of piles (examination at hospital showed no hemorrhoids). He was told to apply cold cream and lemon juice. He went home and took a rectangular jar full of cold cream, unscrewed the top and placed a lemon in the mouth of the jar, and then pushed the jar and lemon up his rectum.[1]

Police investigation revealed that this patient had been guilty of attempted seduction with young girls. We assume, therefore, that this accident occurred during an act of sexual perversion.

[1] Editor's Note: Here again as in the case reported by Dr. Kraker, in this type of case too much credence must not be placed in the patient's history.

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AN UNUSUAL CASE OF MAYHEM

MUTILATION OF EXTERNAL GENITALIA BY ANGRY PARAMOUR

ALBERT S. HARDEN, M.D., F.A.C.S.

Visiting Surgeon, Presbyterian Hospital, Associate Visiting Surgeon, Newark City Hospital

NEWARK, N. J.

THE Crimes Acts of New Jersey[1] describe mayhem as follows:

Any person who, from premeditative design, evinced by lying in wait for the purpose, or in any manner, or with intent to kill, maim or disfigure, shall cut out or disable the tongue, put out an eye, cut off or slit the lip, cut off, slit or destroy a nose or ear, or cut off or disable any limb of another, willfully and on purpose, shall be guilty of a high misdemeanor.

To those of us working in large general hospitals it is not uncommon for the unusual case to appear from time to time; however, in reporting this case we are unable to find any record of a similar attack perpetrated upon a female. For fiendishness and moral depravity it appears to have few parallels.

An investigation made by the police disclosed that the victim, a white woman, thirty years of age, had for the past three years intimately known her paramour, frequently staying at his home. About one year ago she contracted syphilis and in due time infected her lover. At the time of the crime both were under treatment.

On the day of the assault she arrived at his home and complained of not feeling well, removed her hat and lay down upon the bed. Apparently without any provocation he struck her on the left side of the face rendering her unconscious. On recovering consciousness she experienced a severe burning pain over the external genitals. Placing her hand upon the parts she found them covered with blood. As she was too weak to arise from the bed, she attracted the attention of the neighbors by hurling a bottle through the window. This had the desired effect, as several of the neighbors broke into the house. A hurry call was sent to the hospital to which she was quickly removed.

Later the police found two sharp razors in the kitchen and also a considerable portion of the external genitalia lying in a frying pan on the kitchen table (Fig. 1).

FIG. 1. Mayhem: external genitals cut off by angry paramour. Portion of external genitals found, by police, in frying pan on kitchen table. Specimen measured 3 × 3½ inches in size and included larger part of mons veneris, upper parts of labia majora and minora and clitoris. Urethra escaped injury. (Office of Chief Medical Examiner, Essex Co. (Newark), N. J.)

On admission to the hospital, the patient was a well developed female, extremely pale and apparently suffering from loss of blood.

A large pear-shaped avulsion of the pubic region extending from the hair line of the mons veneris including the clitoris and upper one-third of the vulva was found. That portion of the wound over the mons extended down to and exposed the os pubis. In the lower part of the right hypogastrium was a deep cut with a protrusion of several inches of omentum. There was profuse bleeding from both wounds.

[1] Laws of 1908, Chapter 45, Section 112.

The patient on arrival at the hospital was immediately taken to the operating room where bleeding from the wound of the genitalia was controlled by deep sutures of catgut. The abdomen was opened and thoroughly explored for any injuries to the bladder or intestines. As none were found, the abdomen was closed. An intravenous injection of 750 c.c. of saline was given and the patient returned to the ward.

The following day a blood transfusion of 500 c.c. was given. The patient's condition improved rapidly. The wound of the genitalia became infected and was treated with saline irrigations and hot sitz baths. Thirty-two days after the injury, the infection being under control, a plastic repair of the pudendal region was performed.

Under constant saline irrigation a linear incision was made at the margin of the abdominal wound on both sides and the superior border, the granulations removed with a curet and the incisions were undermined for a sufficient distance to allow the wound margins to be approximated. As there was some tension on the sutures, it was deemed advisable to make a few counter incisions parallel to the first incisions and then sew them longitudinally, thus relieving the tension upon the sutures. This procedure was most satisfactory as the wound surfaces approximated with no tension. As there were no labia or clitoris remaining, the same technique was followed to within a centimeter of the urethral orifice and a self retaining catheter inserted. The vaginal wall was then dissected out to the extent of about 0.5 cm. and the adjacent skin, which had evidently been severed but not removed as in the upper portion, was undermined and the two margins were brought together with interrupted silkworm gut sutures to within a centimeter of the urethral opening.

Recovery was more or less uneventful with the exception of a low grade infection around one of the vaginal sutures. This was cleared up by hot sitz baths. The patient was discharged forty days after her admission to the hospital. While from the esthetic viewpoint it left much to be desired, to quote, it was "a couvert."

Unfortunately we have never had the opportunity to follow up this most interesting case as the patient had departed for parts unknown, nor was any arrest made as her lover too vanished. However, a recent police report states that they have become re-united. So as the Bard of Avon says, "all's well that ends well."

INFANTICIDE BY BURIAL

CARMINE G. BERARDINELLI, M.D.

Assistant Medical Examiner, Essex County (Newark), N. J.

NEWARK, N. J.

THE baby shown before this clinic is a female, white infant, six months old. You will notice a perfectly normal, healthy child of this age (Fig. 1).

At the present time the baby is of fair nutrition, is bottle fed, and is being well cared for by the mother.

The unmarried twenty-three year old mother, unattended, gave birth to the baby at home; completely concealing the fact that she was pregnant from her parents.

One morning last January the daughter complained of abdominal pain. Her mother came to my office seeking advice since the girl had previously consulted me over a year ago for severe dysmenorrhea. The mother of the girl stated that her daughter had been free from this symptom for the last nine or ten months. Suspecting a recurrence of the same complaint, I prescribed a similar treatment as before.

In the meantime the girl went into labor. She locked herself in her room. During the period of expulsion of the fetus, she cried so loudly that it came to the attention of her parents. Her father broke into her room and found that a baby had been born and was lying on the floor, apparently lifeless. The girl, during delivery, had been standing at the side of her bed; the baby had dropped on to the wooden floor, breaking the umbilical cord a few inches from the placental implantation. The afterbirth had not been delivered.

In my opinion, the father, thinking that the baby was dead and in order to avoid family disgrace, picked up the infant and carried her to the back yard.

He dug a hole with a spade about one foot deep and placing the naked baby in it, covered it with dry dirt, which was not packed down forcibly. The outdoor temperature at this time was about 30°F. In the meantime, the girl's mother was busy cleaning the room.

After burying the child, the father came to my office and asked me to come over and attend his daughter, whom he stated had had a miscarriage.

When I arrived at the house and saw the conditions, I immediately asked to see the miscarriage since I wished to examine the fetus. The mother stated that the child was some 6 inches in length and was buried in the back yard. In order to ascertain whether the afterbirth had been delivered, I insisted upon seeing it and went out with the father who dug it up carelessly with a spade.

I was astonished to see a baby, not quite full term, weighing about 5 pounds and about 14 inches in length, with about 16 inches of the umbilical cord attached to the navel. The cord was collapsed and its distal extremity plugged with dirt. Though the face was covered with dirt, I found no dirt in the mouth or the nostrils.

On holding the infant up in the cold air, it started to cry feebly, moving both the arms and the legs.

I rushed into the house with the baby, calling for hot water and warm blankets. In the kitchen, I tied the cord, placed the baby in a warm bath and cleaned off the brown dirt clinging to the body. In the meantime, the child began to cry louder and artificial respiration was unnecessary. I then placed the baby in warm blankets.

From the parent's statement, I believe the baby was buried for at least three-quarters of an hour.

The mother and child were rushed to the City Hospital in an ambulance where the mother was delivered of the retained placenta and the child placed under further care. Ten days afterward, following an uneventful recovery, mother and child were sent home, both in excellent physical condition.

During the last six months, I have been called upon to attend the child only three times for slight colds.

In dealing with this case from the legal aspect, it was confidentially reported both to the Medical Examiner and the police.

It was decided to make no charges, pending the outcome of the case. The benefit of any doubt was given to the parents who

Fig. 1. Perfectly normal healthy white female infant, at six months of age. This baby was buried alive after birth for three-quarters of an hour. (Office of Chief Medical Examiner, Essex Co. (Newark), N. J.)

considered the child dead before burial. They were completely exonerated.

Infanticide is the killing of a child at or soon after its birth. The common methods employed in homicidal infanticide are: (1) Suffocation, such as overlaying, pressing the infant's face into the pillow, closing of the nose and mouth with the hands, clothes, etc. (2) Application of external violence, such as dashing head against the floor, wall, or striking the infant with some object, etc. (3) Strangulation by ligature or by the hands. (4) Drowning, especially in toilets, cesspools, sewers, etc. (5) Exposure.

It is distinctly uncommon, in this country, to bury infants alive. Continental medicolegal literature, however, shows that infanticide by this method is by no means rare, and there are numerous reported cases of burial in the ground, ashes, dried leaves, straw, clothing, etc.

It is a well-known fact that often even in normal deliveries, the fetus does not breathe immediately after birth, even though the umbilical cord has been severed. This apneic period in the newborn, between the cessation of placental life and the beginning of the pulmonary one, may last from a few seconds to about two minutes. On rare occasions, the duration of this period of apnea may be several hours. In such cases the child may not be breathing but still be alive.

Numerous experiments on newborn animals and in man have shown that the newborn can stand the total privation of air for a longer period than those who have lived several days.[1] Furthermore, in cases of burial in which recovery has taken place, the pulverulent or dusty condition of the media, in which the body has been buried, permits through its interstices the penetration of a certain quantity of air.[2]

In medicolegal literature many instances of cases similar to this one have been recorded.

Fodere[3] reported 2 cases of newborn infants who were deeply buried by their mothers and then dug up after several hours.

Govard[4] reports the case of a newborn female infant who was bundled up in clothes and buried under a stack of straw and was found alive seven hours later.

Lazzaretti[5] reports the case of an unmarried girl, who soon after being delivered, buried the newborn in the garden of her father, who immediately went to dig up the fetus and found it still living, although it was buried for three-quarters of an hour, with face down and the mouth full of dirt. In this case the baby lived.

Bardinet[6] reported a case of a newborn infant buried in 25 cm. of dirt and dug up after eight hours. The child lived four days.

In occasional instances, therefore, some of these infants buried alive have been resurrected but rarely live for any great length of time.

The case herein reported is one of the few instances in which the child is alive and perfectly healthy six months after being buried alive.

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CASE OF SUPPOSED ASSAULT

JAMES V. JASO, M.D.

Voluntary Assistant, Department of Pathology, Newark City Hospital

NEWARK, N. J.

TO properly interpret and evaluate certain injuries, one must be familiar with and appreciate the habits and customs of various races. The following case well illustrates the importance of this:

FIG. 1. Supposed assault. (Office of Chief Medical Examiner, Essex Co. (Newark), N. J.)

The deceased, a forty-five year old Chinaman, about whose previous medical history nothing is known, walked into a store and fell to the floor without saying a word. When picked up by the ambulance he was still alive but was dead on arrival at the hospital.

On removing his clothes, numerous purplish bruises were seen over his chest anteriorly. Assault being suspected the case was reported to the Medical Examiner's Office.

Autopsy showed a man, forty-five years of age, 5 feet, 5½ inches in height, weight 135 pounds, in fair state of nutrition. Over anterior chest, extending from the clavicles down to the costal margins, there were eight discrete, elongated, somewhat eliptical purplish recent bruises, ranging in size from 1 to 1½ inches. A few of these bruises overlapped. There were no distinct brush abrasions over these bruises and no display of colors as seen in older injuries. On section, these bruises showed very definite hemorrhages in the skin and subcutaneous tissues, not extending into the deep muscles. There were no deep injuries, no fractured ribs or signs of compression of the chest.

The rest of the autopsy showed the left lung collapsed, with extensive chronic acinous tuberculosis and cavitation at the apex. There was a large, hemorrhagic, serofibrinous pleurisy with effusion compressing the lung. The pleural cavity contained a quart of serosanguinous fluid. The right lung was scarred at the apex and contained a cavity 1 inch in diameter with surrounding areas of fibroid phthisis. The other organs were normal except for secondary lesions accompanying an old pulmonary tuberculosis. It was concluded at autopsy that the injuries were superficial in nature and did not cause or influence death which was due to chronic pulmonary tuberculosis.

The police were asked to investigate and ascertain the cause of the bruises. Investigation revealed that the day before death a Chinese friend had repeatedly beaten on the deceased's chest with the back of a large brass spoon. This is an old Chinese remedy for the relief of pain in the chest.

The deceased had complained of pain in his chest, and instead of using heat, or other common forms of counterirritation, they had gone back to a custom centuries old. Interestingly enough, modern medicine is beginning to show added interest in the old forms of treatment; as for example, control of bleeding by the use of prepared snake venom which has also been advocated for the relief of pain in cancer.

Necropsy was performed by Dr. Martland, to whose office I am indebted for the opportunity of reporting this case.

INDUSTRIAL ACCIDENT: HEAD SPLIT WITH ELECTRIC DRYING FAN

JAMES V. JASO, M.D.

Voluntary Assistant, Department of Pathology, Newark City Hospital

NEWARK, N. J.

DR. Martland has taught us for years that a very important function of the Medical Examiner is to prevent wastage of human life in industry. This means not only the investigation of deaths due to industrial accidents, from machinery, etc., but also the recognition of well known and new forms of occupational diseases that may arise from time to time. The discovery of industrial radium poisoning in the watch dial industry and the occurrence of osteogenic sarcoma in these workers, the eradication of benzol poisoning in the artificial leather industry, were all largely accomplished by adherence to the principle of careful investigation of all industrial deaths.

FIG. 1. Industrial accident. Head split by fan. Large electric fan and oven used to dry chemical pigments in a pigment and color works. Workmen were instructed to stop fan before entering drying chamber to remove trays of pigment. Presence of blood and scattered brain tissue on floor of oven may be noted. (Office of Chief Medical Examiner, Essex Co. (Newark), N. J.)

FIG. 2. Same case. Note complete sagittal section of head down to lower lip with complete evisceration of brain. Similar accidents in early days of aviation were caused by cranking propeller. (Office of Chief Medical Examiner, Essex Co. (Newark), N. J.)

It is surprising how this important phase of the Medical Examiner's duties differs in various parts of the country. The usual custom has been to connect the medical examiner's and coroner's work chiefly with the investigation of outright murders, suicides, fatal auto accidents, etc.

The victim in this case was a white male, forty-five years old, killed outright by a large electric drying fan. The head was split sagittally as far as the lower jaw.

He was employed in a chemical pigment and color plant. His work consisted in opening the steel doors of a large drying oven and pulling out frames containing trays of drying chemical pigments. In the corner was a large drying fan, about 6 feet in diameter, which revolved at enormous speed, circulating hot air throughout the oven. The instructions were to always shut off the switch before opening the doors. The switch was located outside the oven. (Fig. 1.)

He was working on a night shift. At about eleven P.M., for some unknown reason, probably because he found that he could do his work more quickly, he entered the oven with the fan still revolving, he having failed to turn off the switch. Another workman found him dead on the floor with his head split. Most of the brain was scattered over the floor and walls of the oven, the cranium being practically empty.

Autopsy showed a male white adult, 5 feet 8 inches in height, 175 pounds in weight.

The skull was completely split almost in the midline, the separation extending through the forehead, face, upper lip, and in back to the posterior border of the foramen magnum, practically bisecting the head in the sagittal section.

The cut through the bone was clean and made practically with one continuous stroke. Only a small amount of brain tissue was left in the skull, about 50 grams, which was sent to the toxicologist for alcohol determination. The cut in front extends down to the lower jaw, the lower front teeth being intact. The bridge-work in the upper teeth was shifted towards the right with very little damage.

In addition to the head injuries, there were large brush abrasions over the left arm and wrist, left knee and right leg.

The right side of the heart contained only a small amount of fluid blood. The other organs showed no evidence of any acute or chronic disease. The stomach contained a small amount of partly digested food in which sweet potatoes could be recognized. There was no alcoholic odor.

Examination of the brain for alcohol showed that the man was not intoxicated.

Necropsy was performed by Dr. Martland to whom I am indebted for the opportunity of reporting this case.

CASE REPORTS

RUPTURE OF THE URINARY BLADDER*

LOUIS BERGER, M.D., F.A.C.S.

Assistant Clinical Professor of Surgery, Long Island College of Medicine

BROOKLYN, N. Y.

THE writer is presenting this case of ruptured urinary bladder with operative recovery for he is at a loss to know how to classify it etiologically.

The literature states that "spontaneous rupture" should be applied to those cases resulting from pathologic changes in the wall of the bladder or to obstructive lesions either at the neck of the bladder or in the urethra, irrespective of the presence of pathologic changes in the bladder. "Traumatic rupture" is applied to those cases in which there is a history and evidence of a penetrating wound. "Concussional rupture" has been proposed for those cases which are due to a fall or blow without evidence of a penetrating wound. The following case does not come under any of these headings.

J. K., a male, aged forty-nine years, was admitted to the Jewish Hospital of Brooklyn.

First admission to hospital December 29, 1932 on the Orthopedic Service.

The patient slipped on the icy street and fell to the ground, striking his right shoulder. He then had pain and noticed that the right shoulder was at a lower level than the left, with some limitation of motion, and a grating sensation over the shoulder when he tried to move the arm. He was treated at the Emergency Room; an *x*-ray taken showed a transverse fracture of the right clavicle at center of its shaft, slight over-riding with outer fragment displaced downward. The shoulder was bandaged and the patient was sent home. He returned to the Clinic five days later and was seen by the attending Orthopedist who advised open reduction of the right clavicle. The patient entered the hospital on the next day, December 29, 1932.

Past personal history. No previous medical illness. Patient had a left hernioplasty performed seven years ago; a nasal operation seven years ago and an operation on his left foot; date and type of operation not stated. The patient denied veneral infection or instrumentation. Has had nocturia two or three times occasionally.

The preoperative urine report was alkaline in reaction, otherwise negative. The temperature, pulse and respiration were normal. Operation was performed December 31, 1932 under gas-oxygen anesthesia. The fracture was corrected with a kangaroo tendon suture, passed through holes drilled in the end of each fragment. The postoperative course was uneventful and patient was discharged January 6, 1933 wearing a figure-of-eight shoulder bandage.

Second admission to the hospital January 8, 1933, sixteen days after injury. The patient stated that on the morning of January 8 he awoke with sharp pain about the umbilicus which remained there for two hours, then migrated to the lower abdomen where it remained for one and one-half hours and then disappeared. The patient vomited once soon after the onset of the pain and had vomited several times since then. He had one enema at home with no result.

Physical Examination: Patient, male, white, appears to be acutely ill. No cyanosis, no jaundice, no dyspnea. Teeth in poor condition, heart soft systolic murmur at the apex. The abdomen showed a generalized distention and

* Read at the regular meeting of the Brooklyn Urological Society.

muscular rigidity throughout. Tenderness, both superficial and deep was present, but seemed more marked over McBurney's point. No masses were palpable. A bandage was still present over the right clavicle. Wound clean and healed by primary union.

The blood count on admission was W.B.C. 15,000 with 92 per cent polymorphonuclears and 8 per cent lymphocytes. No urine examined. The temperature was 100°F. pulse 100 and the respiration 24.

The clinical impression at that time was that we were dealing with a case of general peritonitis due to a perforated viscus, probably a perforated acute appendix but a ruptured duodenal ulcer could not be ruled out definitely for it was impossible to obtain any history different from that noted.

Operation was performed the same day, January 8, at 6 P.M. under spinal anesthesia. A mid-right rectus incision was made which later was extended downward, final length of incision being about 8 inches. On opening peritoneal cavity a serosanginous fluid came out under pressure; about 2 quarts were evacuated; no blood clots noted in the peritoneal cavity. No odor to fluid. No evidence of air or gas escaped. On exploring the abdominal cavity, a rent was found on the superior surface of the bladder which was 2½ inches long, running almost anteroposteriorly. The edges of the rent were clean cut. No evidence of inflammation, malignant disease or diverticula was found in the bladder. The interior of the bladder contained some blood clots, but no other evidence of disease. The prostate was not enlarged. There were no other signs of internal or external injury. The omentum was adherent to the old left inguinal hernia scar at the internal ring and was covering the rent in the bladder. The right kidney was slightly enlarged.

The rent in the bladder was closed with 2 layers of suture: first layer No. 0 chromic including submucuous and muscular layer; second layer of suture, No. 1 chromic, a continuous, non-penetrating peritoneal suture (Lembert). The fluid was evacuated by suction. The omental adhesion was released. A Mikulicz tampon drain was inserted into the rectovesical pouch, covering the bladder and emerging at the lower angle of the wound. The wound was closed in layers. A mushroom self-retaining catheter was passed through the urethra into the bladder.

The laboratory report on the peritoneal fluid was: "The concentration of the urea in this specimen of fluid was sufficiently excessive to indicate the presence of urine."

Being rather surprised with the operative findings, we waited a few days and again questioned the patient. He now told us that since the fall, he had experienced slight burning pain on urination and passed small amounts at a time more frequently since the accident. January 7, 1933, his first day home after the shoulder operation, the patient felt fine. There was no incident of physical strain, violent exercise or trauma of any kind. On January 8 patient awoke about 7:30 A.M., urinated a small amount, no pain. He had his usual mild morning cough due to cigarettes which was no severer than on previous mornings. Immediately following the cough, he experienced severe epigastric pain which doubled him up and was followed by a profuse drenching sweat. He felt like urinating but was unable to urinate or to move his bowels.

January 9, first day postoperative there was a mild abdominal distention, serosanguinous discharge from wound. Bloody fluid draining through catheter. T. 100°F. P. 120. The treatment was the administration of glucose by hypodermoclysis and venoclysis. Consultation was arranged with the genitourinary surgeons.

January 11, third day P.O.T. 101°F., P. 110. General condition better. Patient stopped vomiting. Abdominal wound dry and clean, urethral drainage clearer. Urine examination: specimen loaded with pus cells.

January 13, five days P.O.T. 99°F., P. 90. Slight leakage on dressing. Sutures removed.

January 15, seven days P.O. Urine discharging from lower angle of wound. This area of wound indurated. Some superficial sloughing of fascia. Urethral catheter partially plugged, less drainage.

January 16, eight days P.O. Urethral catheter not draining. Catheter removed and a new indwelling soft catheter inserted. Bladder urine grossly purulent.

January 17, nine days P.O. Mikulicz drain removed. Only slight abdominal drainage.

January 23, fourteen days P.O. Some urine leaking through lower angle of wound. Slight wound infection treated with acriflavine 1:1000. Urethral catheter plugging. Same removed and new one inserted, which was removed in twenty-four hours.

January 26, eighteen days P.O. Abdominal wound dry. Now urinating spontaneously. Urine report shows urine alkaline. Many pus cells in clumps.

February 3, twenty-six days P.O. Patient voiding regularly. Urine clearer. Abdominal wound healing and dry. Patient out of bed and in wheel chair.

February 6, twenty-nine days P.O. Patient walking about. Cystogram taken and reported 2 ounces of residual urine. Examination of bladder reveals a normal outline to the organ.

February 8, thirty-one days P.O. Wound clean, granulating; edges strapped. Patient discharged from hospital.

The patient was treated and dressed in the Out-patient department every other day.

February 25, forty-eight days P.O. Patient is voiding ten times by day and four times by night. Has slight burning on urination. Has residual urine 1 ounce. Bladder irrigated with 1:5000 of acriflavine. Urine reported shows 10–15 W.B.C. and 30–50 R.B.C. per high power field.

March 3, fifty-four days P.O. Patient is better. Nocturia two or three times. Urine is clearer.

March 9, sixty days P.O. Patient feels better. Urinates two or three times by day and two or three times by night. No burning, urine clear.

Patient developed an incisional hernia and he was admitted to my Service at the Jewish Hospital July 24, 1933. A hernioplasty was performed, convalescence was uneventful and patient was discharged August 10, 1933.

Patient was again seen October 20, 1934 and he was perfectly well, complaining of no urinary symptoms.

In conclusion, from the history and findings of this case, one has to speculate as to the cause of the rupture of the bladder. Is it possible for incomplete rupture to occur at the time of injury; namely tearing of all coats except the peritoneum, which held the bladder intact until the sixteenth morning after the injury, and that at that time, after coughing, the peritoneum itself gave way over a full and distended bladder?

WILMS' TUMOR

WILLIAM B. COLEY, M.D., F.A.C.S.

NEW YORK CITY

IN view of the fact that a permanent cure of Wilms' tumor is so extraordinarily rare, I believe that the following case should be of interest:

CASE I. *Wilms' tumor (adenosarcoma) in an infant, treated by nephrectomy and prolonged postoperative treatment with the toxins of B. prodigiosus (Coley toxins). Patient well twenty-nine years later.*

G. D. male, aged eighteen months, weight 21 pounds, had a nephrectomy performed for a large sarcoma of the kidney, on August 27, 1905, by Dr. J. W. Perkins of Kansas City, Mo. *Microscopical diagnosis:* mixed-cell adenosarcoma.

Although the prognosis was regarded as quite hopeless, Dr. Perkins, on my advice, started the patient on a course of prophylactic toxin treatment. This was continued, with occasional intervals of rest, over a period of two years. The doses ranging from ½ to 6 minims, usually produced a temperature of 100–101°F., and sometimes 104–105°F. The patient made a complete recovery, and a letter received from him one week ago stated that he was still in excellent health, more than twenty-nine years later.

I did not appreciate the importance of this case until I reviewed the literature on the results obtained by nephrectomy (above) in the treatment of malignant tumors of the kidney in infants and children, and found only 2 cases in this country well over five years. One of these was Abbe's case published in 1897,[1] and again later, when his patient was well fifteen years after the operation. The other case was cited by Geschickter and Widenhorn in their admirable paper on Nephrogenic Tumors;[2] this was the case of White, in which the patient was well seven years later. According to these authors, 25 followed-up cases of a group of 35 cases of Wilms' tumor in infants and young children treated at Johns Hopkins Hospital, shows no five-year cures; the same is true of a group of 16 cases operated upon at the Presbyterian Hospital in Chicago on the service of Dr. Bevan. In his classical work on "Surgical Diseases of the Kidney and Ureter," Henry Morris reports no five-year cures in his own experience, and states that there are but a very few in the entire literature.

I have had very little experience with Wilms' tumors in infants and children.

I had one case in 1898 in a child aged five years, in which, after trying the toxins for a short while without success, I did a transverse peritoneal operation for a very large tumor of the kidney. No postoperative treatment was given. Metastases developed and the child died within a year. Another case was sent to me from Lincoln, Nebraska, about five years ago, shortly after a nephrectomy had been performed. Prophylactic toxin treatment was started by myself and continued at home by the local physician. In spite of this, a recurrence developed in a few months and proved fatal within a year.

To be of any use, I believe the toxins should be begun as soon as possible after the patient has recovered from the effects of the operation, and continued for a long period of time; this need not seriously interfere with his usual routine of life.

CASE II. Adenocarcinoma of kidney in adult treated by nephrectomy and postoperative treatment with Coley's toxins.[3] This form of treatment was carried out in the case of P. K., male, aged forty-four years, who was referred to me in December, 1928, by Dr. Charles G. Kerley, suffering from a very large tumor of the kidney which had almost reached the inoperable stage. I turned him over to Dr. Barringer who performed a successful nephrectomy. *Microscopical diagnosis:* papillary adenocarcinoma.

The patient was given two exposures of high-voltage *x*-rays at the Memorial Hospital, and was then discharged on January 1, 1929. On January 26, prophylactic toxin treatment was begun by Dr. J. S. Chaffee of Sharon,

[1] *Ann. Surg.*, 25: 360, 1897.

[2] *Am. J. Cancer*, 22: No. 4 (Dec.) 1934.

[3] Through the courtesy of Dr. Benjamin S. Barringer I am permitted to publish the case.

Conn. and continued under my direction over a period of three months. Twenty-seven injections were given, highest dose 10½ min. A letter from the patient this morning states that he is in excellent health over six years later.

I believe we are justified in urging the use of the toxins of erysipelas and Bacillus prodigiosus as a prophylactic after nephrectomy in operable malignant tumors of the kidney when we consider that at least 2 patients with far-advanced inoperable malignant tumor of the kidney in adults, have recovered under toxin treatment alone, one patient remaining well for sixteen years, and the other for twenty-three years, when last traced. Both of these cases were treated by other surgeons who had had no previous experience with the method. They were reported in my paper read before the Third International Cancer Research Conference in Brussels, 1913. The first was treated by Mr. Arthur Connell, Professor of Surgery, University of Sheffield, who sent me the history:

Mrs. M. W., aged thirty-seven years, had two children. In 1908 she first noticed a "lump" in the right side, associated with indefinite pain in the right loin and semilunar line below the costal margin. There was a feeling of constriction of the waist, and occasional attacks of fainting. On two occasions (October, 1910, and August, 1911) blood was noticed in the urine. For a few days prior to her admission to the Royal Infirmary on January 31, 1912, she had passed very dark blood. On February 1, an exploratory operation was performed and a small piece of tissue removed for microscopical examination. According to Prof. Beattie, this suggested a hypernephroma of the kidney. On February 15, 1912, a transperitoneal nephrectomy was performed. Four months later, examination by Mr. Connell revealed a huge recurrent tumor in the right kidney pouch. The patient was readmitted to the hospital, and treatment with Coley's toxins begun at once. She was discharged on October 26, 1912, more than four months later, at which time Mr. Connell reported that no trace of the tumor could be felt, and the patient declared she had never felt better. I saw Mr. Connell in 1928, and he stated that the patient was still well sixteen years after treatment.

The second case was treated by Dr. H. K. McDonald and Dr. A. S. Burns of Halifax, Nova Scotia.

This patient, a female, aged twenty-seven years, had complained of rapidly deteriorating health since the early part of 1912. In August of the same year her physician discovered a large mass in the iliolumbar region. She was brought to Halifax and examined by Dr. McDonald and Dr. John Stewart who decided to perform an exploratory operation. This revealed the lower pole of the kidney to be occupied by a large tumor which was adherent to a much larger growth anteriorly. The patient was then turned upon her back and a laparotomy was made. This disclosed a large retroperitoneal mass which it was impossible to remove. A section of the growth was examined microscopically by Dr. W. A. Lindsay, and pronounced a round-cell sarcoma. The patient made a very poor operative recovery, and was looked upon as an entirely hopeless case. As a last resort she was put upon small doses of Coley's toxins. Dr. McDonald stated that if they were of no benefit, they would perhaps have the other effect and put the patient out of her misery. However, she showed immediate and rapid improvement, and after the eighteenth injection, her physician could find practically no evidence of the tumor remaining. By January, 1913, she had gained 20 pounds in weight, and was able to be up and about the house. The toxins were discontinued on November, 1912, and although the patient was urged to take another course of treatment later, in view of her improved condition, she refused to do so. By June, 1913 she had gained 32 pounds in weight. Fifteen years later, Dr. McDonald performed a hysterectomy for a fibroid tumor; at this time he had an opportunity of examining the whole abdomen, and he stated that he could find no evidence whatever of the tumor of the kidney. A recent letter from him states that the patient remains in excellent condition at the present time, twenty-three years later.

I believe that surgeons will welcome the knowledge that they now have at their command a systemic agent which, used as a routine measure after nephrectomy in malignant kidney tumors, offers definite hope of materially lowering the appalling mortality following operation alone.

OSTEOMYELITIS*

J. J. KIRSCHENMANN, M.D.

Assistant Surgeon & Chief of Surgical Clinic, Norwegian Hospital

BROOKLYN, N. Y.

OSTEOMYELITIS usually means a long drawn out disease with hospitalization, repeated operation and even amputation at times. Since we have been using bacteriophage in the treatment of osteomyelitis we have not found it necessary to amputate a finger or hospitalize a case. Our series in which we have used bacteriophage is very limited, but in so far as it consists of consecutive cases without a failure, we feel that the method of treatment is of some value. In the care of these patients a mixed stock bacteriophage has been used without respect to the causative organism.

The bones involved were: humerus 1; ulna 1; phalanges 6; tibia 2; toe 1.

We have had 6 cases of osteomyelitis of the finger in various stages of bone degeneration. Our treatment has been to incise and evacuate the pus when present and then to inject the phage. Two cubic centimeters of stock phage are injected into the destroyed bone by introducing a hypodermic needle into the area of roughening and then forcing the phage in under considerable pressure. An injection of phage is given every second or third day until there is no discharge from the sinus. At this time the part is *x*-rayed and if the bone shows signs of regenerating, no further phage is used. The part is then *x*-rayed at intervals until complete regeneration has taken place. In addition the patient takes 2 teaspoonsful of cod liver oil with viosterol after meals. We feel that the vitamin D helps the patient to mobilize and use his calcium.

The average disability in osteomyelitis of the phalanges was fifty-six days. This is the time from the beginning of the phage treatment until the wound was completely healed and the patient returned to a gainful occupation. The shortest disability was twenty-four days and the longest 142 days.

CASE REPORTS

CASE 1. C. G., male, aged forty-nine, was seen by us May 10, 1934, complaining of pain in his index finger of several days' duration. There was a scar across the tip of his index finger from an injury received two years ago. The finger tip is swollen to three times its normal size with fluctuation and abscess formation. The abscess was incised. *X*-ray showed an osteomyelitis of the distal phalanx with the lower end entirely destroyed. Six injections of 2 c.c. of stock phage were given into the destroyed bone at intervals of two days. At the end of twenty-four days the finger was normal in size, shape and function and the patient able to return to work. *X*-ray showed complete regeneration of bone after fifty days. (Fig. 1A and B.)

CASE II. P. C., male, aged seventy-one, was seen by us June 18, 1934. He had hurt his finger eight days previously opening a closet door. On examination his right thumb was three times the normal size with pus exuding just back of the nail bed. The bone felt very rough when probed through the sinus. The opening was extended laterally and then connected by rubber drainage with an incision made through the pulp of the finger.

June 20, 1934 *x*-ray showed an osteomyelitis of the distal half of the distal phalanx. Fourteen injections of 2 c.c. each of mixed stock phage were given into the bone through the anterior incision. The patient was able to work at the end of forty-three days at which time function was normal. *X*-ray showed complete regeneration of bone September 17, 1934.

* From the Out-patient Surgical Service, Norwegian Hospital.

Case III. S. A., scrub woman, aged forty-nine, was seen by us December 4, 1933. She stated that she had had a piece of needle in the tip of her third finger for four months, which she had removed one week ago. On examination she had an abscess of the terminal phalanx of the third finger with a lymphangitis extending up the forearm. The abscess was incised and drained.

December 8, 1933 a diagnosis of osteomyelitis was made and verified by *x*-ray.

Fig. 1A. Fig. 1B.

Fig. 1. Case I. A. Osteomyelitis of index finger, before treatment. B. Final results.

December 19, 1933 a daily injection of stock bacteriophage was started. Twenty-five injections in all were given before the *x*-ray showed definite signs of bone regeneration. From this time on the bone continued to regenerate although the superficial tissues were slow in healing due to the patient's poor general condition. At the present time she has a well-formed finger with 90 per cent function at the terminal phalangeal joint. She was able to return to work at the end of 142 days.

X-ray showed complete regeneration at the end of six months.

Case IV. J. B., aged eight, school boy, was seen by us March 30, 1934. An old water boiler had fallen on his finger one-half hour previously. The skin was avulsed anteriorly from over the middle phalanx of the third and fourth fingers. The laceration over the third finger anteriorly extended down into the joint cavity between the middle and terminal phalanx.

Fig. 2A. Fig. 2B.

Fig. 2. Case VII. A. Before treatment with bacteriophage. B. Final result.

April 9, 1934. The anterior tendon sheaths of the third finger were infected with involvement of the lumbrical spaces. *X*-ray showed a beginning osteomyelitis of the middle phalanx. Tendon sheaths and lumbrical spaces were incised and drained.

Fig. 3A. Fig. 3B.

Fig. 3. Case IX. A. Before treatment. B. After phage treatment.

April 13, 1934. Palmar abscess was incised and drained. Ten injections of mixed stock bacteriophage were given into the palmar space, tendon sheaths and into the joint cavity at the site of the osteomyelitis with complete regeneration of bone. Function at the joint was limited to 50 per cent. It was completely healed twenty-five days after the start of the bacteriophage treatment.

Case V. E. A., female, aged five, caught the end of her thumb in an automobile door May 13, 1934, avulsing the nail and soft part and leaving the bone exposed and protruding beyond the soft part. One-quarter inch of bone protruded. *X*-ray showed a fracture through the exposed bone.

May 16, 1934. The finger was markedly swollen and infected with pus exuding around the bone. On May 22, a diagnosis of osteomyelitis was made. Four injections of mixed stock bacteriophage were given into the tissues surrounding the bone. The infection cleared immediately with complete regeneration of bone after fifty-two days.

Case VI. C. B., male aged nine, was seen by us July 19, 1934 after crushing the tip of his fifth finger in a heavy iron door. Examination showed the terminal phalanx of the fifth

right finger to be amputated and dangling from a skin attachment on the inner side. The end of the finger was brought into position with four skin sutures.

July 21, 1934. The end of the finger was dark blue in color due to circulatory failure. All sutures were removed and the patient advised to wrap his hand in an electric pad and apply constant heat.

August 6, 1934. There was definite evidence of osteomyelitis with roughening of the bone and discharge, verified by *x*-ray. The patient was given four injections of stock bacteriophage at two-day intervals.

September 17, 1934. After forty-two days the *x*-ray showed the bone to be completely regenerated and united to the proximal portion with no evidence of osteomyelitis. It was completely healed after fifty-two days.

Case VII. C. M., aged thirteen, school girl complained of severe pain in the right shoulder which developed while carrying fourteen books under the right arm. She stated that her father was tubercular and that she ran an evening temperature of 99.°F. On examination there was marked tenderness over the head of the humerus with independent motion between the head and the shaft. A diagnosis of epiphyseal separation of the head of the humerus was made. *X*-ray was negative for pathology. The arm was immobilized. At the end of one month definite callus was present. *X*-ray at this time showed callus formation, but no definite diagnosis was made. *X*-ray of the chest was negative for tuberculosis. The child ran an evening temperature of 100°F.

December 18, 1933. *X*-ray diagnosis of osteomyelitis was made. The part was not incised or drained. Fourteen injections of 2 c.c. each of stock bacteriophage were given into the diseased bone. At this time the bone showed definite evidence of regeneration. Fifty-nine days from the start of phage treatment there was complete restoration of bone and function. (Fig. 2A, B.)

Case VIII. Mrs. A. M., white, about fifty years of age, fell six weeks previously and compounded both bones of the left forearm just above the wrist. When seen by us November 9, 1933 the original cast was still on. *X*-ray showed a fracture of the lower end of the radius and ulna in poor position.

November 11, 1933. The cast was removed, sutures removed and a large amount of pus exuded from a sinus which ran down between the ends of the fractured ulna. There was no union between the ends of the ulna.

December 2, 1933. *X*-ray showed an osteomyelitis of the fractured ends of the ulna with an absorption of bone. Twelve injections of 2 c.c. each of stock phage were given through the sinus into the space between the fragments of the ulna.

February 17, 1934. Seventy-seven days after the start of the phage treatment the *x*-ray showed complete union and clearance of the osteomyelitis. Function at this time was 60 per cent.

Case IX. Female, white, aged thirty-three, was first seen by us November 26, 1934. She had had a compound fracture of the tibia and fibula in April, 1934 for which she was treated at another hospital. When seen by us she had a slight bowing of the left leg with two sinuses over the upper third of the tibia. The upper sinus was surrounded by an ulcer 1 inch in diameter from which granulations exuded. Each sinus led down into an area of roughened bone. The foot was markedly swollen with 25 per cent function at the ankle. *X*-ray showed an old united fracture of the tibia and fibula with a defect in the upper part of the lower outer side of the tibia.

December 1, 1934. A piece of sequestrum 2 inches long and 1 inch wide was removed from the upper sinus. Bacteriophage was started.

January 12, 1935. A piece of sequestrum ½ inch in diameter was removed from the upper sinus. Fourteen injections in all of stock phage were given into the upper sinus.

March 12, 1935. *X*-ray shows an old fracture of the tibia and fibula with the defect entirely filled in. No further evidence of osteomyelitis.

At the present time the patient is well except for a small eroded skin area over the tibia. Her function at the ankle is 70 per cent. *X*-ray showed complete regeneration of bone after one hundred and two days. (Fig. 3A, B.)

Case X. T. M., male, white, floor scraper, was first seen by us July 31, 1934. On January 20, 1934, he received a compound fracture of the left leg for which he was treated at another hospital. When seen by us his left leg and foot were swollen to two times normal size. The lower portion of the leg was markedly discolored. There were three excoriated areas

over the crest of the tibia through which sinuses led down into roughened bone.

Diagnosis: chronic osteomyelitis of the tibia.

X-ray showed an old fracture of the lower third of tibia and fibula. There was external displacement of the upper fragment of the tibia and fibula one-half the width of the bone. Considerable callus was present. A small spur projected from the lower anterior part of the upper fragment. There was an osteomyelitis of the fractured end of the lower tibial portion. Thirty-three injections of 2 c.c. each of stock bacteriophage were given into the bone through the sinuses at intervals of two or three days.

October 17, 1934. A piece of sequestrum 1 inch long was removed from a sinus.

December 17, 1934. A piece of sequestrum 1 inch long was removed from a sinus.

December 15, 1934. A large abscess of the leg was incised and drained.

February 11, 1935. *X*-ray showed complete regeneration of bone with no evidence of osteomyelitis.

At present the bone is completely restored and the sinuses healed. The skin over the lower one-third of the leg is excoriated due to circulatory stasis. *X*-ray showed complete regeneration of bone one hundred and sixty-five days after the start of phage treatment.

Case xi. A. J., male, white, aged sixty-four, was first seen by us July 13, 1934. He had had pain in the big toe of his left foot for three months. The toe had been incised previously and had been draining since. On examination the big toe was markedly swollen with a sinus ½ inch in diameter discharging pus. The sinus went down into an area of roughened bone and the toe felt as though it had been fractured.

X-ray showed osteomyelitis of the proximal and distal phalanx of the big toe.

He was given twenty-nine injections of stock bacteriophage at one or two day intervals into the diseased bone. Sixty-six days after the first phage treatment the bone seemed to be completely regenerated and the wound healed. The patient never returned for his final *x*-ray, but we are informed that his toe has remained healed since.

CONCLUSION

Eleven definite cases of osteomyelitis involving five different bones have been presented. All of these have responded to phage treatment as outlined. Cases i, ii, iii and xi, we would previously have treated by amputation. We regret that these cases were not cultured before treatment was started.

PROTECTION OF A BODY CAST IN AN INFANT

David Kuperstein, M.D.

Assistant in Obstetrics and Gynecology, Greenpoint Hospital

Brooklyn, N. Y.

THE protection of a body cast in an infant has been troublesome to the infant, the surgeon, and the parents. Because of soiling, numerous applications of the cast necessitate frequent manipulation of the part to be immobilized.

CASE REPORT

A male infant, four months old, having a congenital scoliosis, was placed in a cast to correct the deformity. A coating of shellac was applied to the external surface of the cast, but after one week, because of soiling, the cast had to be removed. The second cast was covered with oiled silk, and the edges secured with adhesive, but because of mold formation, this had to be replaced at the end of two weeks.

When a third cast was applied, water-proof adhesive was used, and by depressing the skin beneath the cast, the adhesive portion was applied to the inner surface of the cast and continued on to the outer surface beyond the area of soiling. This cast remained in situ for four weeks, and was removed only because the infant outgrew it. The two succeeding casts which were applied remained dry for four and five weeks respectively. After removal, the inside of the cast was inspected and found to be dry.

RUPTURE OF THE QUADRICEPS TENDON*

DAVID SLOANE, M.D. AND MARIAN FRAUENTHAL SLOANE, M.D.

Adjunct Orthopedic Surgeon, Hospital for Joint Disease

Assistant Adjunct Orthopedic Surgeon, Hospital for Joint Disease, O. P. D.

NEW YORK CITY

SUDDEN muscular pull rarely ruptures the powerful quadriceps tendon of the thigh. When this does occur, however, there ensues a chain of disabling symotoms which necessitates early operative surgery. The following illustrates the sequence of events in such cases.

CASE REPORT

P. C., white male, aged fifty years, engineer by occupation. While pushing a wheelbarrow the patient's left foot slipped forward and he fell to the ground. He was unable to arise. I saw him about fifteen minutes after the accident. At that time he complained of severe pain in his left knee and inability to straighten his leg or stand. His knee was markedly swollen with a definite hollow just above the patella. The anterior surface of the lower femur was readily palpated in the depth of this hollow. Active extension of the knee was nil. Immediate *x*-ray examination ruled out a fractured patella and thereby confirmed my diagnosis of a rupture of the quadriceps tendon. He was immobilized in plaster, with the knee in full extension, at my office, because he refused hospitalization. Two days later he was admitted to the Hospital for Joint Diseases with the understanding that the operation was to be performed under local anesthesia.

Following novocaine infiltration, the suprapatellar region was exposed by a longitudinal incision. The quadriceps tendon was completely torn across about 1 inch above its insertion into the patella. The tear extended laterally and medially into the fibrous expansions of the vasti. The suprapatellar pouch of the quadriceps was open, and the knee joint proper was filled with blood and clots. After removing the blood from the knee joint, the torn ends of the tendon, including the lateral expansions, were approximated and united by interrupted chromic mattress sutures. The superficial tissues were then closed and the knee fixed in full extension by means of a circular plaster cast.

Three weeks postoperatively the cast was bivalved and physiotherapy instituted. One month postoperatively weight-bearing was begun and after four months the patient returned to work.

Examination five months after his return to work revealed a painless full range of knee motion.

DEFINITION

A rupture of the quadriceps tendon is a tear of that tendon produced by a muscular action.

INCIDENCE

The condition is much rarer than generally suspected. Sonnenschein[1] reported only 4 cases at the Harlem Hospital from 1922–1927. There have been no cases of this type for the past five years on either Dr. Frauenthal's orthopedic service at the Hospital for Joint Diseases or Dr. Crossman's surgical service at the New York City Hospital. Gallie[2] considers this type of injury fairly common, but even he saw only 6 cases over a two year period.

ETIOLOGY

The immediate producing cause of a ruptured quadriceps tendon is a sudden violent contraction of the muscle with the knee fixed in flexion. This same mechanism may produce a fracture of the patella. In young people the quadriceps is stronger than the patella, but after the fifth decade the quadriceps tendon gives way. This is

* Presented at the Hospital for Joint Diseases.

especially apt to occur if the quadriceps has been weakened by fibrosis due to an old injury, lues, arteriosclerosis, or excessive obesity.

DIAGNOSIS

There is a history of a misstep followed by a sudden violent attempt to straighten the flexed knee. The patient has a sharp pain above the knee and falls to the ground. He is unable to arise without assistance or to bear weight on the injured limb.

Examination reveals a huge swelling of the knee joint with a characteristic hollow just above the patella. Palpation in the depths of this hollow outlines the anterior surface of the lower femur. Voluntary extension of the knee is impossible although passive motion is present. *X*-rays help in ruling out a fractured patella.

TREATMENT

There can be no compromise as to treatment. Early suture of the ruptured tendon is the only procedure that offers a strong useful limb. Rest in bed, plaster, or the use of splints may give some return of function due to partial healing by scar tissue, but the patient is left with a weak and unsteady extremity. He has difficulty particularly in climbing up and down stairs. He has periodic attacks of pain and swelling which keep him bedridden.

Delay in operating allows retraction and shortening of the tendon ends. This necessitates the use of fascial grafts which complicate the operative procedure and prolong the after-care. The results of the delayed operation, while good, do not compare with the result of immediate operation.

The disadvantages of delayed operation are seen in Milch's case.[3] The patient came to him two years after the original injury. At operation he found that the ends of the torn tendon had undergond extensive fatty degeneration. When this mass of fatty tissue was removed there was a 10 inch gap between the patella and the normal muscle.

Wishner[4] had a similar experience. His patient came to him six months after the accident complaining of difficulty in going up and down stairs. His limb was unsteady, he fell frequently, and he had periodic attacks of a painful swollen knee joint. At operation a peculiar condition was noted. The synovial membrane of the knee, lining the quadriceps pouch, had overgrown the patella so that the patella itself was covered front and back with synovial lining. As in Milch's case,[3] considerable fascial plastic surgery was necessary. Although the results in both these patients were excellent, they required extensive surgery and would have undoubtedly been better off if operated upon immediately.

In my case the tendon was repaired about forty-eight hours after the original injury. The operative procedure consisted of simple chromic suture of the torn tendon. The end-result was a painless knee with complete range of motion. The patient is an engineer in one of our large theatres and his work necessitates considerable kneeling and carrying of heavy loads. He has been back at active work for the past six months. His excellent functional result may very well be ascribed to early operation.

SUMMARY

A man over forty missteps, falls to the ground and is unable to arise. Inability to extend his knee, a swollen joint with a definite hollow above the patella, and a negative *x*-ray make the diagnosis of a ruptured quadriceps tendon. Immediate open operation is the only rational procedure.

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TRAUMATIC RUPTURE OF THE BICEPS BRACHII

HORACE STIMSON, M.D.

CHICAGO

INTRODUCTION

THIS paper is written from the point of view of the specialist in traumatic surgery. My interest in this particular injury began with an instance in 1929, was stimulated by a second case in 1931 and by a third in 1933, in the course of the examination and treatment of injured persons. The 3 are the only ones discovered in the series of approximately 10,000 injured, or supposedly injured, persons examined and treated by us in the past six years. The third case came on August 22, 1933, and was under our postoperative care in October when I heard Gilcreest[1] of San Francisco read his excellent paper on this subject before the Clinical Congress of the American College of Surgeons in Chicago. His analysis is so thorough, covering 100 studied cases, and his review of the literature is so complete that I do not propose to cover the ground he has done so well, but to add the 3 following cases to the list of those he reported. It is to be recommended that all physicians, particularly surgeons specializing in the handling of injuries, should read his paper with care. The first 2 cases I describe briefly, the third in more detail.

CASE I. A muscular white laborer of middle age while lifting a heavy object and in the position of partial flexion of the elbow felt a sudden snap and pain in the upper arm, followed by arm weakness and immediate disability, and he noticed a peculiar bunched appearance in the biceps muscle. His employer sent him at once to our office, where I examined him and found the recognizable signs of a rupture of the biceps muscle: an undue prominence of the outer belly of the biceps in its lower half, and a visible and palpable tender sulcus between this bunched muscle and the lower anterior deltoid edge, an increase in the deformity on having him flex the elbow against resistance, a tenderness on pressure deep under the anterior portion of the deltoid, but none high at the shoulder, weakness in performing elbow flexion and forearm supination: ecchymosis beginning about the anterior middle portion of the upper arm. I diagnosed rupture of the long head of the biceps, probably occurring just at the musculotendinous junction. This man refused operation, received heat and later massage treatments and muscle training, was disabled about six weeks, and recovered with a painless arm, a moderate weakness in flexion of the elbow and supination of the forearm (compared with the other arm), and of course his biceps deformity remained, with some muscle atrophy and a persistent hiatus in the outer belly of the biceps.

CASE II. 1931. A muscular white laborer of middle age was injured in much the same way, by straining in lifting a heavy object, with the elbow partly flexed. He presented himself to the office the same day as injured, with the same complaints as listed in the preceding case. Examination showed an obvious rupture of the belly of the outer portion of the biceps brachii. The examiner's finger could be inserted in this transverse defect, which was tender, filled with soft hematoma, and the outlines of the retracted bulging muscle edges could be seen and felt above and below the defect; the gap widened on having him flex the elbow against resistance with forearm supinated, and this increased his pain. There was no shoulder tenderness, swelling or ecchymosis. I operated the following morning through a 4 inch upper arm anterolateral skin incision and found the aponeurosis of the outer belly of the biceps ruptured completely and separated, with the muscle itself ruptured transversely, but the muscle sheath and sub-

[1] Gilcreest, E. L. The common syndrome of rupture, dislocation and elongation of the long head of the biceps brachii, an analysis of one hundred cases. *Surg. Gynec. Obst.*, 58: 2A, 322–340 (Feb. 15), 1934.

stance were perfectly healthy in appearance, indicating that the indirect trauma alone was responsible for the rupture. The muscle portions were found separated about 1½ inches, the gap extended down to the humerus, and was filled with fresh hematoma, with some blood extravasation subcutaneously outside the sheath. I flexed the elbow to relax the biceps, evacuated the blood clot, approximated the muscle with deep mattress sutures of No. 2 chromic catgut, approximated the bulging muscle edges with other interrupted sutures, and repaired the sheath smoothly over the sutured muscle. The arm was dressed in almost full elbow flexion and forearm pronation, the wound healed per primam, skin sutures were removed about the tenth day; passive motion was started about three weeks postoperative, active motion and heat lamp and massage started five weeks postoperative, and he was discharged to his labor about eight weeks postoperative. The motion, strength and contour of the repaired muscle were fully restored, but the thickened cicatrix in the repaired muscle sheath could be palpated easily. There was no apparent muscle degeneration or atrophy above or below the suture line.

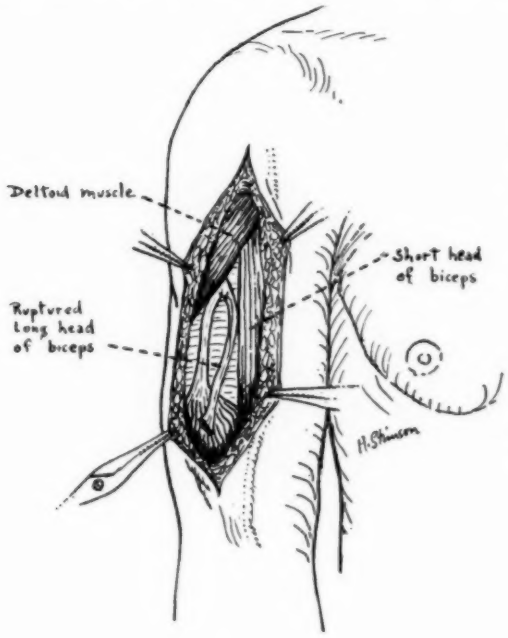

Fig. 1.

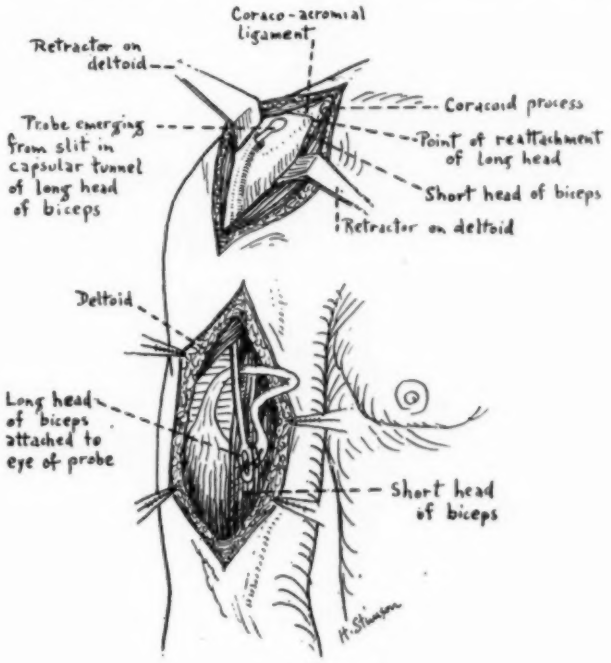
Coraco-acromial ligament
Retractor on deltoid
Coracoid process
Probe emerging from slit in capsular tunnel of long head of biceps
Point of reattachment of long head
Short head of biceps
Retractor on deltoid
Deltoid
Long head of biceps attached to eye of probe
Short head of biceps
H. Stimson

Fig. 2.

Case III. R. B., negro, aged twenty-nine, was injured August 22, 1933, 3:45 P. M. while doing stevedore work, loading a boat. He was lifting a bale of rubber and in so doing he twisted his right arm near the shoulder, felt something "pop" in his arm, was immediately disabled, noticed a peculiar bunch of muscle in his upper arm, reported his injury at once to the superintendent and was sent to our office where I examined him two hours after the injury. He was found to be a young, healthy and very well muscled negro (see photographs). He denied any previous injury or trouble with this arm. He exhibited a distal retraction and undue prominence of the outer belly of the right biceps, a visible and palpable gap proximal to this and up as far as the anterior

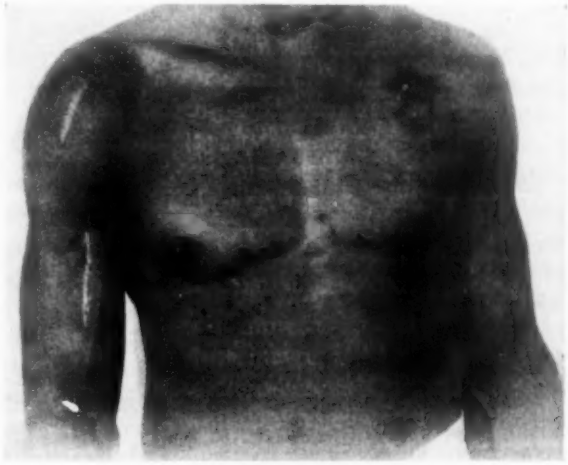

Fig. 3.

deltoid fold, a hematoma palpable in the gap, tenderness of this part of the biceps, also tenderness of very slight amount at the front of the shoulder joint. The retracted outer belly of the biceps was prominent but rounded, which made me think the tendon of the long head of the biceps was ruptured somewhere between the musculotendinous junction and the glenoid attachment, and that the muscle belly itself was not ruptured. On having him flex his elbow against resistance, the muscle deformity and the gap increased, the muscle bunch sliding distally, and he complained of a catching pain in the biceps region.

Fig. 4.

Fig. 5.

Operative Procedure: He consented readily to an operation, which I performed the following morning, meanwhile packing the arm in ice caps overnight. Anesthetic: Ethylene with ether sequence, to obtain full muscle relaxation. First, a 3 inch longitudinal skin incision was made anterolateral at the middle third of the upper arm, the outer belly of biceps was found shortened and bunched up, with the muscle surface transversely wrinkled and relaxed and pulled away from the inner belly of the biceps; the long head had evidently snapped with violence as I found it looped backward in the muscle gap and lying free in the midst of the hematoma which filled this hollow (See Fig. 1). The tendon was healthy, glistening, white, smooth and strong, and examination of the free end showed that it had torn completely loose from its cartilaginous attachment at the glenoid rim, called the supraglenoid tuberosity.

Since the subsequent operative procedure which I followed differs somewhat from that reported previously in the literature, it is described in detail, assuming that the anatomy of the upper arm and shoulder is well known to the reader. The elbow was flexed to relax the biceps. To save a long upward extension of the original incision, a second incision 4½ inches long was made over the anterior portion of the deltoid, its fibers split and retracted, exposing the shoulder joint capsule in the portion containing the groove or tunnel for the long head of the biceps. A properly curved long probe was passed up the tendon sheath, from

the emergence under the anterior border of the deltoid to a point high on the exposed shoulder capsule, a ⅓ inch longitudinal slit was made in the capsule for the point of the probe to emerge, the end of the tendon was sewed to the eye of the probe, then probe and attached tendon were threaded up through the sheath and through the capsular slit. Care was taken not to cut through the deep layer of the capsule which would have unnecessarily opened the shoulder joint synovia. The end of the tendon was then sewed strongly with No. 1 chromic gut to the coraco-acromial ligament close under its attachment to the coracoid process, and near the lateral fibers of the short head of the biceps. Several reinforcing sutures were taken between the tendon and capsule at its new emergence from the capsule slit. (See Fig. 2.)

I would advise the surgeon undertaking such a repair to pass the probe in a retrograde fashion downward from a slit in the capsular bicipital groove if he cannot readily pass the probe upward from under the anterior deltoid fold as I did. Also I would suggest his using fine braided or twisted silk in the reattachment of the tendon, to avoid the danger of suture absorption and pulling loose in the succeeding four to six weeks before the tissue union is firm, also that he should strongly reinforce his suture of the tendon to the coracoid process or by lateral interrupted sutures to the tendon of the short head of the biceps, or to the coraco-acromial ligament for added strength.

The foregoing procedure immediately restored the normal contour and tenseness of the outer belly of the biceps. The skin was closed in the usual manner with a waxed silk continuous suture.

Progress and End-result: He was discharged from the hospital forty-eight hours postoperative, with the arm in a sling and with the elbow in moderately acute flexion. The skin sutures were removed on the eighth day. He cooperated well in abduction shoulder exercises for the deltoid, and two weeks postoperative could fully abduct the shoulder; the sling was removed at the end of three weeks and the elbow exercised; heat lamp and massage treatments were started at the twenty-sixth day, and nineteen such treatments given at intervals of two to four days, the last treatment being October 30, 1933, approximately nine weeks postoperative. Meanwhile he had returned to his regular stevedore work on October 19, about seven and a half weeks postoperative, returning for a few final treatments for his complaint of slight stiffness and pain in the shoulder (none in the arm region).

Fig. 6.

He came in for a follow-up examination and photographs in February, 1934. The accompanying plates (Figs. 3–6) show very well the functional and anatomical result obtained, as well as the location of the scars. The deltoid scar measured 4½ inches, the upper arm scar 3¼ inches; there was no muscle deformity in any position, or stress on the arm. He had a full range of motion at all joints in comparing the two arms and no loss of strength. Measurements were as follows: Circumference of the right or injured arm over the biceps (extended) 10¾ inches, left 10¾; over biceps (flexed) right, 12½, left, 12¼; over large part of forearm, right, 10¼, left, 10¾. He is right-handed.

REMARKS

These 3 cases illustrate the fact that has already been pointed out by Gilcreest, Loos, Keen and others, that the long head of the biceps brachii can and does rupture simply from indirect trauma, namely a sudden strong unbalanced muscular effort,

exerted by the biceps, and that this can occur with healthy muscle and tendon, and in comparatively young individuals. All three of the patients we treated had exceptionally strong muscular development and were engaged in heavy labor as a regular occupation. As Dr. Paul Magnuson pointed out in his discussion of Dr. Gilcreest's last paper, this injury is one which is probably unrecognized, misdiagnosed or not even looked for by many of us. It is not so rare an injury that it should be taken lightly. It is important from an industrial standpoint, as it causes partial or complete disability for heavy labor; it is subject to gratifying results when recognized and surgically repaired immediately after the injury occurs. It can occur in young, healthy, strong individuals, and the structures which rupture can be healthy tissues, thus suitable for surgical repair.

Personally, I have not seen the type of slow or spontaneous rupture of the long head of the biceps brachii which Gilcreest, Meyer and others have described, occurring from the underlying cause of degenerative changes in muscle and tendon, myositis, arthritic roughening at the shoulder with consequent slow attrition of the tendon, and final stretching or tearing. These types should be carefully distinguished from the acute traumatic rupture, as Gilcreest says, so that the examining physician may clearly understand and point out whether the condition is truly compensable, from the industrial standpoint, as caused by injury (direct or indirect violence); or whether the rupture is spontaneous, the result of a disease process, gradual in development, and not the result of a single violence. The careful diagnosis is also very important in determining whether or not to operate for there can be much less hope of surgical success in dealing with diseased tissue than with healthy tissue.

It should be pointed out that the second case cited had the combination of aponeurosis and muscle rupture, which is rare as reported in the literature, the aponeurosis seldom tearing. I also want to recommend to the consideration of surgeons the operative procedure described in the third case, for it restores the original course of the long head of the biceps through its capsular tunnel in a truly anatomical manner, as compared with the method favored by Gilcreest of disregarding the capsular groove and suturing the tendon to the coracoid process or to the tendon of the short head of the biceps. Gilcreest apparently leaves the tendon entirely out of its old bed in the capsular groove. Naturally the success of the procedure I recommend depends on the finding of a healthy tendon which has simply been detached by trauma from its origin at the glenoid tubercle. The writer agrees with Gilcreest that an intracapsular reattachment is hazardous and unjustifiable, as there is no need to invade the shoulder synovia.

SUMMARY

Traumatic rupture of the long head of the biceps brachii muscle by indirect force is illustrated by 3 industrial cases; 2 were operated on, and details of the operative procedure given in the third case, with recommendation of a new method of reattachment of the avulsed tendon.

In closing, I wish to thank Dr. C. R. G. Forrester and Dr. A. H. Mason, my associates, for their aid in the preparation of this paper.

SYMPATHICO-BLASTOMA

OF THE SECOND DORSAL SYMPATHETIC GANGLION

MILTON SILLS LLOYD, M.D.

Assistant Thoracic Surgeon, Sea View Hospital

NEW YORK CITY

THE patient was a white male, aged forty-three, admitted to St. Vincent's Hospital, S. I., May 5, 1934, T. 99.6°F., P. 100, R. 21.

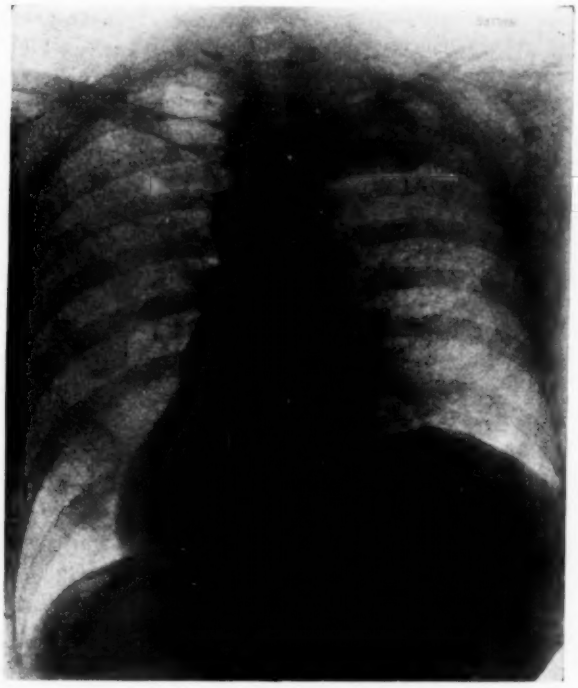

FIG. 1. *X*-ray of chest showing shadow of tumor mass occupying right apical region. Right diaphragm is high although paralysis of phrenic nerve was not demonstrated.

History. Two months previously the patient had a "cold" which cleared up, leaving a dry cough and an aching and burning pain in the right posterior chest, becoming gradually more severe. He had lost 12 pounds of weight.

Physical Examination. There was a bulging in the right posterior chest from the first to the fourth dorsal spines, and the *x*-ray confirmed the suspicion of a tumor mass occupying this area, as shown in Figure 1. (All *x*-rays by Dr. M. B. Radding.)

Laboratory examination revealed nothing of importance as a positive finding except a gradually falling red blood cell count and a gradually rising white blood cell count.

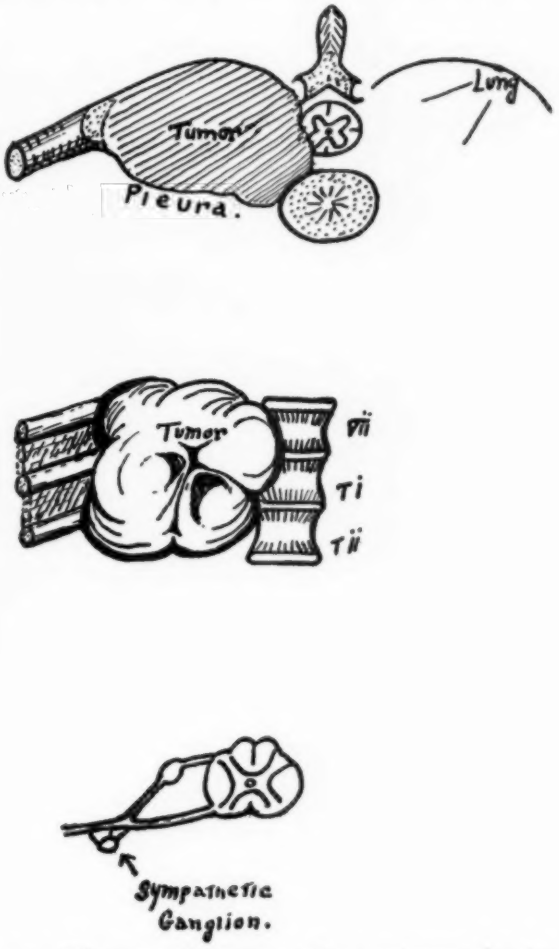

FIG. 2. Diagrams of tumor made at autopsy, showing anatomical location and mechanism of compression of cord by transgressing space between pedicles of transverse processes.

Bronchoscopy, performed on June 8, 1934 was negative, except for a slight bulging inward of the right side of the trachea.

* Read before the New York Society for Thoracic Surgery.

Diagnostic pneumothorax failed to permit of thoracoscopic examination because of pleural adhesions.

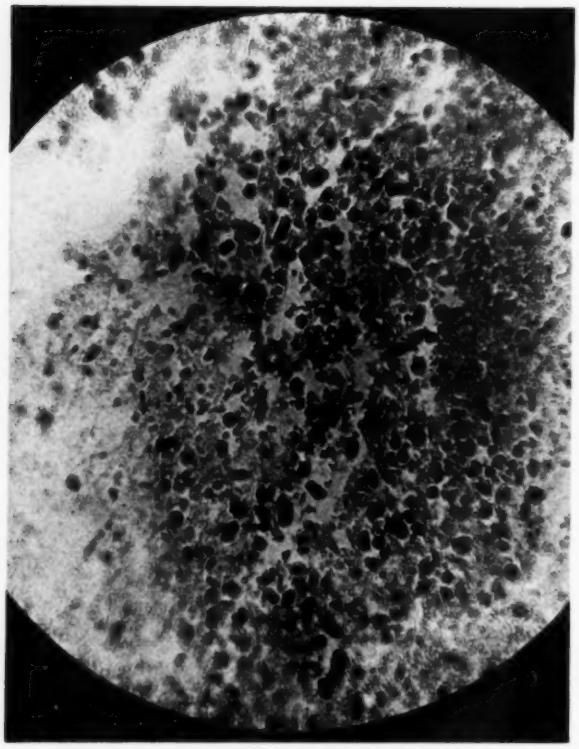

Fig. 3. Microscopic section of tumor. (× 625.)

Clinical Course of Disease. Shortly after admission the patient began to complain of a sense of "pins and needles" on the right side of his face and neck. This gradually turned into a clammy feeling of the skin with alternating sensations of heat and cold. The oculocardiac sympathetic reflex was so marked at this time that pressure on the eyeballs or the carotid sheaths almost stopped the heart.

On May 20, the patient complained of perspiring freely on the left side of his face, while the right side of his face was dry. It was also noted at this time that the legs were cold and clammy. This was followed by a gradually increasing weakness of the lower limbs, and by the beginning of June the patient was unable to leave his bed.

Horner's syndrome began to appear and later became very apparent.

On June 14, control of the urinary sphincter muscle was lost and the patient had to be regularly catheterized from that date on. At the same time, there was an exaggerated left knee jerk and a suggestion of a Babinski on the left side.

Neurological consultation, with Dr. C. C. Hare on June 15, showed evidence of an early compression of the cord at the level of the third dorsal vertebra, and by June 23, further examination of the nervous system by Dr. S. Brock showed the lesion to have produced a complete transverse myelitis. It was interesting to note that the paralytic symptoms occurred on the left side first, although compression occurred from the right.

The surgical diagnosis on this case at the time, was ganglion neuroma of the second dorsal sympathetic. There was however, naturally, a difference of opinion in the case of such rare occurrence and operation was not recommended.

The patient gradually became worse and died on July 25, with complete incontinence, complete paralysis of the lower body and severe cystitis and hydroureters, and hydronephrosis.

X-ray examinations showed no bone involvement of the vertebrae or ribs.

Autopsy Report. (Excerpt) (Drs. M. Freund and H. Sussman). "After removal of all organs in the upper portion of the right chest, an orange sized round, flat tumor bulged into the chest cavity, covered by the parietal pleura, which was not infiltrated. Lying on the second and third ribs, near to the vertebral junction, this tumor was elastic in consistency and infiltrated through the intervertebral openings, into the spinal channel at the height of the second and third dorsal vertebrae.

Microscopically, there could be recognized very large polygonal cells which formed a characteristic part of the tumor; they had vesicular nuclei. The protoplasm showed frequently one, occasionally two processes. These cells lay within a fibrillar matrix, they could be identified with reasonable certainty as ganglion cells. In addition, there were present sheets of small cells resembling neuroblastic cells. Diagnosis, sympathicoblastoma." There were no metastases.

The writer is indebted to Drs. H. H. Cochrane and J. S. Diamond for their permission to report this case, and to Dr. H. Angrist for the preparation of the photomicrographs.

CARCINOMA OF CERVICAL STUMP*

LOUIS E. PHANEUF, M.D., SC.D. (HON.)

BOSTON, MASS.

MRS. A. Mc, forty-one years of age, consulted me on October 19, 1928. She complained of bleeding freely and irregularly from the vagina. On July 25, 1928, approximately three months previously, she had had a supracervical hysterectomy for fibroids, performed in a neighboring state. The pathologist had discovered carcinoma on the cut surface of the cervix and had so reported. Her only other complaint was backache, which had existed for some time. Her menstruation was established when she was fourteen years of age, the periods had been regular, of the twenty-eight day type, had lasted six to seven days, pain had been but rarely present and she had occasionally passed large blood clots. This patient had been married seventeen years and had had six children, five of whom were living; the oldest child was sixteen and the youngest two years of age. She had had no miscarriages. The first two labors were terminated instrumentally, the others were normal, as were the puerperia.

The physical examination revealed no abnormalities except for the pelvic condition. The abdomen showed a recently healed median pelvic incision. The vaginal examination presented a deep laceration of the perineum and relaxation of the vaginal walls, accompanied by a cystocele and a rectocele. An everting carcinoma of the cervical stump, involving the entire cervix and extending to the right vaginal vault was discovered; this bled freely when touched and sponged with cotton. The rectal examination confirmed these findings but did not add to them. She was referred to the Carney Hospital for radium and deep *x*-ray therapy.

On October 25, 1928, under general anesthesia, a biopsy was taken from the cervix and .050 mg. of heavily screened radium element was placed against the cervix and left for twenty-four hours, giving a dose of 1200 millicurie hours. It was impossible, at the time of this radium application, to find the cervical canal. The biopsy specimen was examined by Dr. Frank B. Mallory, who reported: "Specimen consists of several pieces of cervix. Diagnosis: Epidermoid carcinoma."

On December 1, 1928, it was found that the cervix had practically healed and that it was freely movable. There was a little granulation tissue at what should have been the external os. A biopsy was taken in this area. It was possible to dilate the cervical canal and to introduce within it .050 mg. of heavily screened radium element, which was left in for twenty-four hours, giving an additional dose of 1200 millicurie hours. The pathological report showed chronic inflammation.

On January 5, 1929, the cervix was found to be healed except for a small sloughing area in the center. The cervical canal was again dilated and .050 mg. of heavily screened radium element introduced in it; this was left in for twelve hours, giving an additional dose of 600 millicurie hours.

On February 9, 1929, without anesthesia, sloughing material was curetted away from a crater in the center of the cervix and .050 mg. of heavily screened radium element placed in this crater and left in for twenty-four hours, a radium dose of 1200 millicurie hours.

On October 25, 1929, one year after the first radium application, the cervix was found to be completely healed and movable; .050 mg. of heavily screened radium element was placed transversely across the cervix and left for twelve hours, a radium dose of 600 millicurie hours.

Radium Dose

October 25, 1928,	1200 mc. hrs.
December 1, 1928,	1200 mc. hrs.
January 5, 1929,	600 mc. hrs.
February 9, 1929,	1200 mc. hrs.
October 25, 1929,	600 mc. hrs.
Total Dose—	4800 mc. hrs.

* Read before the Obstetrical Society of Boston.

During the course of this year the patient was given deep *x*-ray therapy by the radiologist at the Carney Hospital. She was then exam.ned every few months until five years had elapsed; she is now reporting once a year and has been instructed to present herself for examination at the slightest sign of bleeding. She was last examined on April 28, 1934, five and one-half years after the initial radium application. She had gained between 40 and 50 pounds in weight and her general condition was excellent. The vaginal examination showed atrophy of the external genitals and vagina, there was a small stump of cervix, the size of the tip of the little finger, high in the vault and the cul-de-sacs were all supple. The rectal examination confirmed these findings and showed that the parametria were soft, not tender and not involved. Her only complaint is an occasional backache when she over-does. She has been heard from recently and is enjoying good health.

The points of interest in this case are:

1. The failure to make a diagnosis of cervical cancer which existed at the time of the supracervical hysterectomy.

2. An apparent cure or arrest in a woman treated with radium and *x*-rays, the initial treatment having been given six years and five months ago.

3. The increased danger of vesicovaginal and rectovaginal fistula when treating with radium, the absence of the uterus removing a heavy source of filtration of the radioactive substance.

COMMENT

During the last twenty years I have seen 8 cases of cervical stump carcinoma. Five of the women were seen over ten years ago, during the course of one year. The sixth forms the basis of the present case report and the seventh and eighth were seen within one week of each other, in March, 1935. Radiation, by means of radium and *x*-ray, was employed. The first five women all died within one year of the t me the diagnosis was established. The sixth, treated by the same method, is well today, six years and five months after the initial radium treatment was instituted. The seventh and eighth have just begun treatment. In 2 of the patients, cases six and eight, I am convinced that carcinoma existed at the time the supravaginal hysterectomy was performed; in the other 6 I cannot be certain.

LEFT PARADUODENAL HERNIA

TWO CASES, ONE WITH RUPTURE THROUGH THE WALL OF THE HERNIAL SAC

CLARENCE F. BALL, M.D., F.A.C.S.

Surgeon, Rutland Hospital

RUTLAND, VT.

PARADUODENAL herniae are not uncommon. However, when encountered they are of sufficient importance to be reported. If there is any variation from the conventional findings these should be discussed as completely as possible.

That a left paraduodenal hernia may not have blood vessels in its ring is denied by some. It is also claimed that all such herniae must have an entering and an emergent loop of gut passing through the opening. This is undoubtedly true of the right paraduodenal herniae, but in the left, the entering loop may come through the wall of the sac so posteriorally as to be wholly within the sac in so far as its orifice is concerned (Naubauer). In such a case only an emergent loop presents itself to the surgeon at operation. Variations in the location of the inferior mesenteric blood vessels in relation to the duodenojejunal fossa allows of a left paraduodenal hernial formation without blood vessels being a part of the orifice or of the anterior portion of the walls of the sac as will be discussed later.

Recently, Andrews,[1] Copenhaver,[2] Desjardins,[3] Dowdle,[4] Exner[5] and others have fully reviewed the literature on the so-called paraduodenal herniae.

A rupture through a retroperitoneal sac is unreported in so far as the author has been able to determine and is perhaps the most unique feature of the first case reported. It was the immediate factor in bringing this case to operation. The second one was an uncomplicating finding in a patient with simple appendicitis wherein the annular ring did contain the inferior mesenteric blood vessels and the walls of the sac were vascular.

CASE 1. The patient was admitted to the hospital 10:45 P.M., complaining of acute abdominal distress. He had had abdominal distress only a day or two previous to his admission. There had been some vomiting and some obstipation.

Examination: (Fig. 1.) Patient was a short stocky boy, six years of age, hands and feet rather broad with thick fingers and toes, suggestive of mild pituitary disturbance. His genitals were only slightly developed. His abdomen was protruberant, tense, and very tender on pressure. It was not as board like as often encountered in some acutely involved abdominal conditions. He could not lie with his legs extended. He was not tender over his kidney areas. The liver and the spleen could not be palpated. No definite mass or masses were palpable through his tense fat abdominal wall. His reflexes were normal. His temperature was 99.8°F., pulse 115, respiration 22. A blood count showed 16,000 whites with 90 per cent polymorphonuclears. His urine, with a sp. gr. 1.030, showed sugar and albumin, with a large amount of acetone. There were a few red blood cells and a few pus cells, also many hyalin casts.

A working diagnosis was made of a general peritonitis complicating an acute appendicitis.

Operative Findings: The abdomen was opened through a right rectus incision with displacement mesially of the right rectus muscle before opening through the peritoneum. A small amount of bloody colored fluid was first encountered on opening the peritoneal cavity. The examining fingers encountered a mass in the mid abdomen not connected with the appendix. (Fig. 2.) The appendix was essentially normal. It was removed by cautery section and the cecum replaced and the wound enlarged

upward in order to better determine the pathology of the mass felt in the central abdomen.

Fig. 1.

Through the enlarged opening a purplish loop of small gut was seen to protrude through a slit in a grayish fibrous sac. No small gut, other than this protruding loop, was visible. This sac was found to contain the entire length of the small bowel with the exception of a few inches of the distal ileum adjacent to the cecum. The hand was passed all around this pouch or sac, and no small bowel was encountered anywhere else in the abdomen. There was a free space between the sac and all portions of the colon and mesocolon. The sac did not fall quite down to the bladder. There was more free space below than elsewhere about the sac. The pouch or sac seemed to be composed of a fibrous fold, or folds, of peritoneum, arising from the lower posterior margin of the transverse mesocolon at its mid portion and surrounding the exit of the duodenum

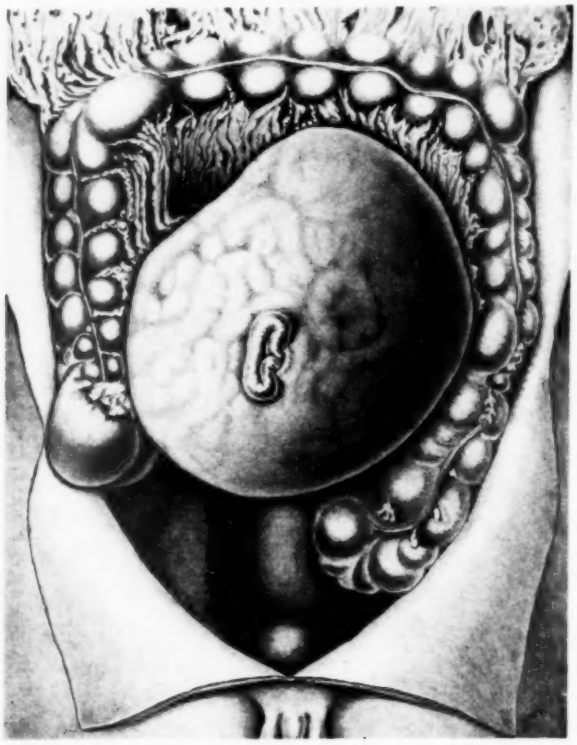

Fig. 2.

from behind the stomach at about the location of the ligament of Treitz.

The index finger of the examining hand was passed along the side of the distal ileum up and into the opening in the sac. This opening pointed to the right and was just below the foramen of Winslow. The opening would admit only two fingers. The opening contained the distal end of the ileum with its mesentery passing out and then downward to the cecum, as the only structure palpable therein. There was no evident constriction of the ileum or its mesentery. There was no entering loop of bowel discernible. There were no arteries or veins in the anterior walls of the sac, or about the opening. No effort to find the blood vessel relations at the base of the sac to the left was attempted, as the patient's condition was too critical to allow of a too extended examination. Since I was not able to palpate any blood vessels in the anterior wall of the sac or in the anterior edge of the orifice, an incision was made upward and dorsalward on the right lateral surface from the hernial slit to and through the ring. (Fig. 3.) There was a result-

ant immediate release not only of the strangulated loop of bowel, but a complete freeing of all of the previously incased small bowel. The folds of the jejunum and ileum then began to assume their normal relations within the abdomen. As this occurred, the walls of the incised sac fell downward and backward and to the left, completely disappearing behind the freed folds of the small bowel. The cut edges of the sac did not bleed. There were no vessels in the orifice to be clamped.

The mesentery to the small bowel did not seem abnormal. The strangulated loop reacted satisfactorily to *hot* towels, accordingly was not excised. There was the usual amount of vascular dilatation throughout the intestinal serosa generally found in inflammatory obstructive conditions. Because of the considerable distention of the small bowel, but more particularly to relieve tension on the strangulated loop, an ileostomy was done with catheter drainage. The wound was then closed with through and through stay sutures.

The patient was in the operating room from 12 midnight, until 1:30 A.M., and was returned to bed in the children's ward in a critical condition. The patient reacted to the operation the following afternoon and evening, with a mounting temperature up to 105.4°F. at 8 o'clock, with a pulse of 140–150, and respirations 28–30. Thereafter temperature, pulse and respiration came down by lysis, to near normal points shortly before the patient's discharge.

His condition thereafter was complicated by wound sepsis, only as associated with the ileostomy fistula. Spontaneous closure of the fistula followed in due course. This patient left the hospital fifty days following the operation, with the wound practically closed. Home dressings, by a visiting nurse, completed the recovery. No abdominal manifestations of a complicating pathology have been present since his operation.

Case II. Mrs. D. had the clinical picture of a mildly acute appendicitis. On opening the abdomen a moderately acutely inflamed appendix was found and excised. In bringing up the appendix, the ileum was found to run into a sac. This sac was found to contain all the small bowel. The orifice which pointed to the right contained blood vessels which coursed over the anterior walls of the sac. No entering loop of bowel was encountered. There was no evident inflammation of the ileum entering the sac. The hand was passed about this sac freely. Its base was at the duodeno-jejunal junction. It was recognized as a left paraduodenal hernia without symptoms, and was left undisturbed.

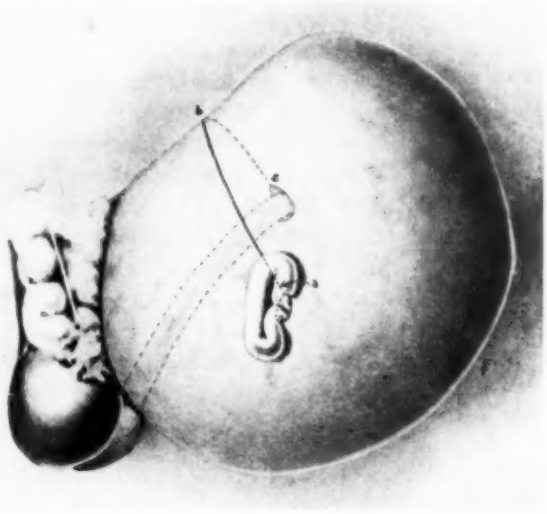

Fig. 3.

The abdomen was closed without drainage. The patient made an uneventful recovery and has had no complicating symptoms since.

DISCUSSION

Moynihan,[6] in his monograph on retroperitoneal herniae, briefly reviews the historical background relative to a study of the various herniae in this location. He states that Hensing in 1742 was the first to mention them, and Huschke in 1844 was the first to describe them, but it remained for Treitz in 1857 to go into their detailed description, as we know retroperitoneal herniae now.

Quoting Moynihan for an authoritative description:

It was he (Treitz) who first recognized that such herniae occur in fossa which are normal and it was he who first gave us any intelligent description of the fossa and their mode of origin. He says: "If in a body with a normal peritoneum one lifts up the great omentum and the transverse colon, and pushes over to the right the mass of small intestines, there will be seen on the left side of the duodeno-jejunal flexure a peritoneal fold. This fold varies in shape and size. Most frequently it is semi-

lunar, the thin concave edge looking upwards and to the right and surrounding the bowel at the level of the flexure. The upper horn of this semilunar fold is blended with the inferior layer of the transverse mesocolon, and especially at the point where the inferior mesenteric vein passes beneath the pancreas. The larger lower horn is continuous on the inner side with the peritoneal investment of the duodenum, and at the outer end with the peritoneum of the transverse and descending mesocolon. In the upper horn, at a variable distance from the edge, lies the inferior mesenteric vein, forming an arch, with the convexity looking upwards and to the left. The lower horn is less distinct, composed exclusively of two layers of peritoneum, and, at some distance from its free border, one sees the inferior mesenteric artery and its branch, the left colic artery. From the relative positions of these two vessels there results a vascular arch (the "arch of Treitz"), which surrounds the fold in question. Behind this peritoneal fold, between it and the duodenum, there exists necessarily a depression or pocket in the form of a funnel, the summit of which is directed towards the duodenum. The orifice of the entry is semilunar, limited on the right by the intestines, the flexure duodenojejunalis; on the left by the free border of the fold. This fossa is in general situated on the left side of the third lumbar vertebra."

Wenzel Gruber, in 1859 and 1861, in describing the fossa of Treitz, but more especially the vascular arch of Treitz, found it sometimes in front of the fossa in the anterior wall, *sometimes behind*, and sometimes distant on the outer wall.

Eppinger in 1870 reported a variation in the arrangement of the vessels in 25 cases examined. In 7 cases the vessels ran in the edge of the fold, in 15 cases the vessels were distant, 4–17 mm. from the edge, and in 3 *cases the vein lay behind the posterior wall of the fossa.*

CONCLUSION

The author believes that the hernial sac encountered in his first case was one of a left paraduodenal hernia, with the vascular arch of Treitz posterior to the duodenojejunal fossa, rather than anterior. Its location, its orifice, and its contents support this view. Its avascularity was due to the unusual location of the inferior mesenteric vessels. I believe this case is more readily explained upon Moynihan's fossa theory, than upon Andrew's developmental rotational theory. The distinctive feature, however, is that of a strangulating hernia through a slit in the wall of a retroperitoneal hernia. A hernia of a hernia!

The second case conformed to the more conventional type of left paraduodenal hernia.

Both cases, similar as to gross characteristics, were surgical findings in operations for clinical appendicitis. Both patients have been perfectly well for over two years.

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OVARIAN CARCINOMATA SIMULATING ACUTE SURGICAL CONDITIONS OF THE ABDOMEN*

PHINEAS BERNSTEIN, M.D.

NEW YORK CITY

OVARIAN carcinomata with symptomatology of acute pelvic or abdominal conditions occur infrequently. Among 64 cases of malignant ovarian growths at Mt. Sinai Hospital there were 4 such cases. The diagnoses in these cases were, respectively, acute suppurative appendicitis, pelvic abscess, intestinal obstruction due to rectal carcinoma and twisted ovarian cyst. Each patient presented a syndrome of pain, nausea, vomiting, chills, fever, abdominal rigidity, leucocytosis and rapid sedimentation time. The literature contains little reference to this subject. The rarity of this atypical clinical onset and the high incidence of incorrect diagnosis in this type of case are of sufficient importance to warrant special attention.

CASE I. T. B., No. 329045. Ovarian carcinoma simulating acute suppurative appendicitis and later pelvic abscess.

A Russian-born housewife was admitted August 12, 1931. She was aged sixty-one years, menopause for eighteen years. An ectopic gravidity had been removed twenty-nine years previous.

Sixteen months prior to this admission she entered another hospital for severe abdominal cramps, pain, vomiting, chills and fever. A right tubal abscess and a subacutely inflamed appendix were removed. For three months thereafter an abdominal sinus persisted. When the discharge from the sinus abated, the patient developed chills, fever and mild abdominal cramps. Upon one of these occasions, the same surgeon rehospitalized her and made a diagnosis of pelvic abscess. A posterior colpotomy was then performed. This drained continuously for the subsequent nine months.

She came to this hospital because of sudden onset of abdominal pain, cramps, nausea, vomiting, chills and fever. There was also marked urinary frequency.

Examination revealed a mass filling the entire pelvis, extending to the umbilical level on the left side and to the anterior superior spine on the right side. A profuse drainage from the colpotomy opening and moderate cervical discharge were noted. The liver was slightly enlarged, irregular, nodular and hard. Additional significant findings were the sedimentation time of eight minutes, temperature 101°–104°F. and 47 per cent hemoglobin with moderate leucocytosis.

The diagnosis was made simply upon examination of pus obtained from the opening in the posterior fornix. Carcinoma cells, hair particles, amorphous calcium elements, leucocytes and debris were identified microscopically. The diagnosis of ovarian carcinoma developed in a dermoid cyst was later confirmed by autopsy.

CASE II. M. F., No. 275008. Ovarian carcinoma simulating pelvic abscess.

The patient, aged thirty-eight years, re-entered the hospital December 25, 1927 after leaving against advice three months previously. Upon this admission she complained of sudden onset of severe, sharp lower abdominal pain, radiating to the back and right buttock with vomiting, chills and fever. There was rebound tenderness and abdominal rigidity.

Bimanual examination revealed a mass extending from one pelvic wall to the other. Areas of softening were palpable at several points behind the cervix. A profuse vaginal discharge and a bloody purulent rectal discharge were noted. Leucocytosis, fever ranging from 101° to 104.4°F. and sedimentation rates of nine and fourteen minutes were observed. The cervical smears for gonococci and the Wassermann test were both negative. Upon

* From the Gynecological Service of Mount Sinai Hospital. Read before the Section of Gynecology and Obstetrics, N. Y. Academy of Medicine.

this admission, as on the previous occasion, the diagnosis of pelvic abscess was made. A posterior colpotomy was performed and pus was obtained.

Microscopic examination of the fluid obtained from the posterior colpotomy opening revealed carcinoma cells. Subsequent exploratory laparotomy disclosed an inoperable carcinoma of the ovary with multiple adhesions to the intestines.

Had not an indication for colpotomy developed, it is unlikely that the correct diagnosis would have been made.

In both these cases the importance of microscopic examination of pelvic exudates is forcefully illustrated.

Case III. D. G., No. 358292. Ovarian carcinoma simulating acute intestinal obstruction.

A forty-eight-year-old housewife was admitted December 12, 1932. Since an appendectomy at the age of fourteen years, followed six months later by a laparotomy for adhesions, the patient had been in excellent health. Suddenly on the day of admission the patient developed severe abdominal cramps, vomiting, distention, constipation, chills and fever.

Examination revealed two lower abdominal scars. The right lower quadrant was spastic and tender. On vaginal palpation a pelvic mass was found filling the cul-de-sac and extending laterally almost to the pelvic walls.

Rectally a crater-shaped, friable, infiltrating mass, almost completely annular, was felt along the anterior wall. Biopsy from this tissue revealed papillary adenocarcinoma, the origin of which was considered to be the rectum.

The patient was, therefore, transferred to the surgical division. At operation an extensive bilateral ovarian carcinoma, densely adherent to the small bowel, was found. The anterior rectal wall was markedly invaded. The ovarian carcinomata were removed together with the adherent and invaded portions of small bowel, and an ileosigmoidostomy and colostomy were performed.

The infiltrating character of ovarian carcinomata, producing symptoms of misleading nature is shown by this case.

Case IV. R. E., No. 369799. Ovarian malignancy simulating ovarian cyst with twisted pedicle.

A forty-year-old unmarried patient was admitted August 13, 1934 with the complaint of vaginal bleeding of three weeks' duration.

She was operated on at another hospital one year previously, after the diagnosis of acute appendicitis and peritonitis had been made, and a chronically inflamed appendix was removed. Three days after discharge she returned with symptoms of intestinal obstruction for which an ileostomy was performed. Three months later she was again admitted to the same hospital with signs of an acute surgical abdomen. The diagnosis of twisted ovarian cyst was made. Immediate operation was advised but the patient left the hospital contrary to advice.

Two months later she was admitted to the surgical ward of this hospital, where a diagnosis of twisted ovarian cyst was again made. Gynecologists called in consultation were of the opinion that the disturbance was not of pelvic origin. However, operation on the surgical side revealed an adenocarcinoma of the left ovary with adhesions to the contiguous intestine.

Fifteen radiotherapy treatments, given after operation, produced an amenorrhea, which in six months was interrupted by an episode of constant and increasing spotting, for which she was re-admitted to the gynecological service of this hospital. Specular examination revealed a smooth, soft, friable rounded mass arising from and occluding the cervix. The bleeding was profuse. Microscopic examination revealed a squamous-cell carcinoma of the cervix.

This case is of added interest because of the coincidence of ovarian adenocarcinoma and squamous-cell carcinoma of the cervix.

SUMMARY

1. Ovarian carcinomata may first manifest themselves clinically with the general picture of an acute surgical abdomen.

2. The symptoms are due to secondary changes resulting from the tumor: infection, adhesions and metastasis.

3. A persistent discharge from a posterior colpotomy opening should suggest carcinoma of the ovary as a possible etiology and warrants microscopic examination.

NEW INSTRUMENTS

CYSTOSCOPE AND TEST TUBE HOLDERS*

PETER JOHN RIABOFF, M.D.

NEW YORK CITY

THE need of a good cystoscope holder is self-evident. It relieves the operator or his assistant from the tiresome

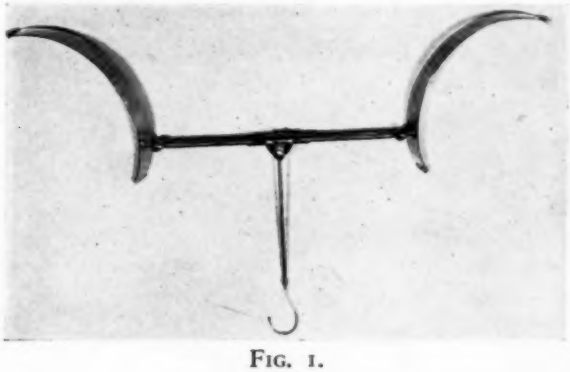

FIG. 1.

holding of the instrument. This is particularly desirable when the excretion of the

FIG. 2.

urine is slow. In the case of female patients, it is practically a necessity.

The unpopularity of all the cystoscope holders offered up to the present time is due to the following causes:

(a) They were heavy

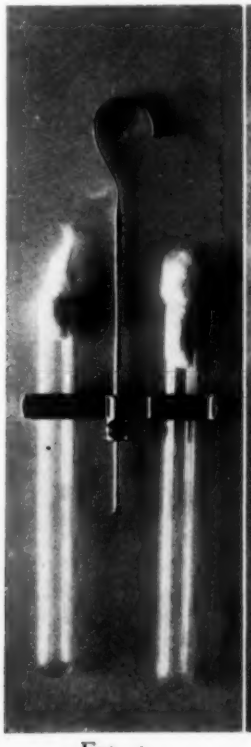

FIG. 3.

FIG. 4.

FIG. 5.

(b) Huge in construction
(c) Hard to manipulate
(d) There was the hazard of injury to or even piercing the bladder because of lack of flexibility.

The object in the construction of the new instrument was to eliminate all these drawbacks and to make a cystoscope

* From the Department of Urology, Cornell University, New York Hospital. Read before American Urological Assn., New York Branch.

holder to comply with the following requirements:

(a) Extremely light
(b) Essentially simple in construction
(c) Easy to manipulate
(d) Entirely safe.

The instrument (Fig. 1) consists of two curved metal bands which rest on the patient's thighs. Two metal rods are attached to these bands, which in turn slide in metal tubes. This arrangement permits the rods and tubes to telescope together. Easy adjustment to the patient (Fig. 2) is accomplished by lengthening or shortening the distance between the bands. A thin rod with a movable hook is attached to the center of the tube. The cystoscope is placed on this hook. The rod moves back and forth; up and down; sways left and right. This motion of the rod in three planes permits the cystoscope to assume a natural position to the urethra. Because of this arrangement it gives more comfort to the patient.

The lack of security of the test tubes suspended and held on the catheter by the cotton plugs led us to devise new test tube holders. There is a holder for one or two tubes. One consists of a rack for two tubes, (Fig. 3) movable on a rod, which is hooked to the cystoscope. The single one (Figs. 4 and 5) has a rack for one tube and a clamp to grasp the catheter.

I wish to acknowledge my appreciation to Mr. Frederick Wappler for the splendid execution of the cystoscope and the test tube holders.

A CEREBELLAR OUTRIGGER

JEFFERSON BROWDER, M.D.

BROOKLYN, N. Y.

THE available types of mechanical devices for supporting a patient upon whom a posterior cranial fossa or upper spinal cord operation is being performed, fail to meet all of the requirements when the entire procedure is conducted under local anesthesia. Although every surgeon develops a technique which best suits his particular needs, it seems that there are unnecessary technical handicaps resulting from the use of a rigid cerebellar outrigger. In an attempt to overcome some of the difficulties encountered during the opening and closing of a "cerebellar" wound in particular, an apparatus has been constructed so that the head and cervical spine may be held in a flexed or extended position as the occasion demands.

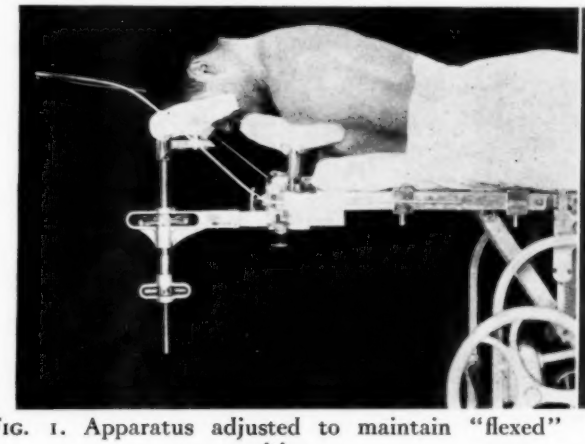

FIG. 1. Apparatus adjusted to maintain "flexed" position.

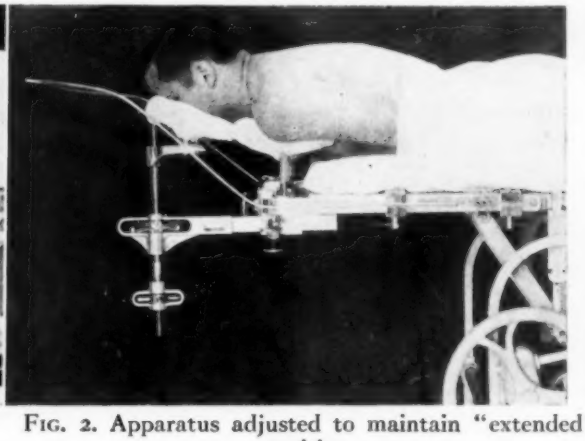

FIG. 2. Apparatus adjusted to maintain "extended" position.

By tilting the headrest as in Figure 1, the contents of the posterior cranial fossa may be easily exposed without cumbersome retractors. When the wound is to be closed, the divided, deep cervical fascia can be approximated without tension, by tilting the headrest as in Figure 2.

For exposing the cervical spinal cord, the flexed position (Fig. 1) is desirable, during the removal of a tumor the extended position (Fig. 2) relaxes the spinal cord and then the flexed position (Fig. 1) may again be obtained, which accurately approximates the wound for closure. This "outrigger" is so constructed that it can be adjusted for any size subject and can be easily attached to any type of operating table.

CHRISTOPHER WIDMER.

CHRISTOPHER WIDMER is a never-to-be-forgotten name in the early history of Toronto and throughout Canada.

He was born at High Wycombe, Buckinghamshire, England, on May 15, 1780.

We learn from the records of the War Office, London, that he was hospital mate in June, 1804 but about a year later (Aug. 1805) he was appointed assistant surgeon of the 14th Dragoons, and surgeon to the same regiment on October 24, 1811. Widmer served with honor in the Peninsular War of 1808–14 and took active part in five important battles, earning him the right to wear the medal with five clasps.

In the War of 1812, with the defeat of Napoleon, England was able to send some assistance to her Colony, and so we find Widmer with some of the 14th Dragoons in Canada. At the close of the war he decided to stay in the Province, so, on February 25, 1817, he was retired on half pay and began the exhausting duties that would fall to one who was the only qualified medical man settled at York (Toronto). Naturally, his fame as a physician and surgeon soon spread and his practice took him on long journeys, often in the dead of winter through almost impassable roads. Only an experienced horseman could have ridden 160 miles without stopping, except to change horses, to attend Lord Sydenham, then at Kingston.

In 1820 one of his ambitions was realized. A hospital was built. It was enlarged in 1824 and again in 1832. In 1832 students attending the hospital for clinical instruction formed the "York Students' Medical Society." In 1835 the medical profession held a meeting at the hospital to form a medical library.

Widmer was interested in public health, at that time at a low estate, and urged daily employment of scavengers.

Widmer accepted many positions and to them gave faithful service. As a member of the Legislative Council he made many journeys to Montreal and Quebec. He was the first president of the Medical and Chirurgical Society, which he founded in 1833. He was one of the first in Upper Canada to recognize the value of vaccination, and had notices published in the papers stating that he would inoculate children of the poor free of charge.

The first medical journal published in Toronto was the *Upper Canada Journal of Medical, Surgical and Physical Science*, 1851–52. In the first volume Widmer's name appears at the head of a number of signatures protesting a proposed new medical bill.

We learn he was a citizen of high ideals, one who had the courage of his convictions. He acquired fame and wealth, and held the foremost place as surgeon of his time in Canada.

In the new home of the old Toronto General Hospital hangs a fine painting of him.

He had one son to whom he was devoted. This son died in 1857, and a year later, while paying a visit to his grave, Christopher Widmer fainted and shortly afterwards died.

T. S. W.

CHRISTOPHER WIDMER

[1780-1858]

The American Journal of Surgery
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[From Fernelius' Universa Medicina, Geneva, 1679.]

BOOKSHELF BROWSING

HUMANISM AND PANTAGRUELISM*

JOHN M. TAYLOR, M.D.

BROOKLYN, N. Y.

THESE are not two new diseases. Humanism and Pantagruelism affected the personality and writings of a noted Frenchman, four hundred years ago. Whether they constituted a pathological syndrome may be open to controversy.

On a previous occasion, to illustrate the influence of the physician in the realm of letters and of culture, I presented a sketch of Sir Thomas Browne, distinguished author of "Religio Medici." About a hundred years before the publication of that classic, there was published in France, a rollicking, humerous satire, entitled "Gargantua and Pantagruel." The author was François Rabelais. A translation in the seventeenth century by Sir Thomas Urquhart, an English interpreter, with a genius akin to that of Rabelias, himself, made of this work a great English classic. The adjectives, Gargantuan and Rabelaisian, are in daily use. The work is still carried on the shelves of modern bookstores and attracts the modern reader.

Though known as a unique personality in literature, it is not often recalled that Rabelais was a French physician. This fact and a realization that this very year marks the four hundredth anniversary of the publication of "Gargantua and Pantagruel," suggested the presentation of the life and times of Rabelais from the viewpoint of the physician of today. A recent biography by Jean Plattard of the University of Poitiers serves as the basis for the following details of his career.

His father, Antoine Rabelais, was a lawyer in the town of Chinon, about twenty-five miles south of Tours. Besides a large town house, he owned a nearby farm and country residence, "La Diviniére." It was here that François Rabelais was born, probably in 1494.

It is well to orient ourselves with the spirit of the times in Europe, when this boy was born. Columbus had just discovered a New World. The Revival of Learning and the Renaissance of Art had already reached their apogee in Italy. Lorenzo, the Magnificent, had just passed away in Florence. Michael Angelo, Leonardo Da Vinci, and Raphael were at the height of their powers. Roderigo Borgia (Alexander VI) was the Supreme Pontiff and, in principle at least, as vicegerent of God, was the ruler of Europe and the New World. His son, Caesar, had rounded out the Papal States. A few years previously the art of printing

*Read at a meeting of the Professional Staff of the Brooklyn Hospital.

by movable types had been invented. The Fall of Constantinople forty years before had sent many Greek scholars into exile throughout Europe to promote the revival of scholarship, that was spreading from Italy to the North.

During Rabelais' life span of sixty-seven years great changes took place. In this era, the chief interpreter to the peoples of Northern Europe of the great intellectual movement of the fifteenth century, was Erasmus of Rotterdam, critic, friend and advisor to the Vatican and to all the Courts of Europe. From the new Scholarship arose the so-called Humanist Movement, which sponsored, among other things, a widespread enthusiasm for a reorganization of the practices, teachings and dogmas of the Church in harmony with the new knowledge.

Continually increasing political friction between the Vatican and the temporal powers of the States of Europe was reaching a climax and impasse. To compromise these controversies and conflicts was beyond the powers of the polished, cultured Erasmus. It fell to the lot of his contemporary, the fanatic, the orator and monk, Martin Luther, to arouse a combination of the common people and the disaffected Princes in a revolt, that brought to an end the Holy Roman Empire and started Europe on the road to nationalism. Latin became displaced as the universal, supranational language of scholarship and religion and there started the development of national literatures.

The times of Rabelais were "the twilight of the age of chivalry and romantic love; the beginning of the age of discovery, the rise of nationalism and the breakdown of the hierarchy of the Church."

Born in this era of controversy and conflict, Rabelais passed his childhood in the country atmosphere between the little town of Chinon and the hamlet of "La Diviniére." He received his early schooling at the neighboring Benedictine Abbey of Saint Pierre du Seuilly. At about the age of fifteen he became a novice in the Franciscan monastery of La Baumette at Angers, some eighty miles west of Tours, the home of his mother's family.

Here, among his fellow students, Rabelais made friends who in later life were to prove influential and helpful, especially Geoffrey d'Estissac, who was to become Bishop of Maillezais, and the duBalley brothers, who later attained eminence and power as Papal ambassadors, soldiers and cardinals.

Either entirely here at this monastery, or probably partly also at the University of Angers, Rabelais pursued his school life until he was twenty-five. Unfortunately, we have no contemporaneous biography of Rabelais and he has left no autobiographical record of his aspirations at this period. For this youth, who had evinced unusual aptitude and zeal for study, now enters the Franciscan monastery of Fontenay-le-Comte to take the vow of poverty of that order and eventually to be ordained to the priesthood. It is true that, in times past, this order had given to the Church such great thinkers as Roger Bacon and Duns Scotus. But in Rabelais' time this Order of the Minor Friars was denounced by the intelligentsia as a group of most ignorant monks, who held it to be heretical to seek too much after knowledge.

We are left to assume that Rabelais apprehended in the Franciscan life nothing which might check his appetite for knowledge, which was becoming his most striking characteristic. He found a kindred spirit here named Pierre Amy, who had smuggled into the monastery a number of books in Greek, some Hebrew textbooks, and some books on Roman Law and Arabic. The two managed to study these, undetected for a considerable time. As an illustration of this Humanist movement in France, this urge for attainment of old and new sources of knowledge, these young men conceived the idea of writing to the great Humanist scholar, Budé, who was secretary to King Francis I. He encouraged and spurred them on to great accomplishments in scholarship. Among evidences of Rabelais' youthful erudition is the fact

that, while here, he translated the second book of Herodotus from the Greek into Latin!

In the town of Fontenay these young monks formed part of a coterie of the most intelligent men for discussing literature, poetry, art and philosophy and all the controversial topics of the time.

Eventually these young Franciscans were disciplined for their studious ardor; for they were detected, and the Faculty of Theology at the University of Paris (the Sorbonne) had solemnly and stupidly decreed the study of Greek to be heretical.

As a consequence, Rabelais soon after appealed to his old schoolmate, Geoffrey d'Estissac, who had risen rapidly in the world and now lived in a palace at Ligugé near Poitiers. Here, as Bishop of Maillezais, he also presided over a Benedictine Abbey. He effected the transfer in 1524 of Rabelais from the Franciscan to the Benedictine order, appointed him as a canon and made him his private secretary.

In his capacity as a member of the Bishop's household and his secretary he found continuing opportunities for study and on his rounds with the prelate enlarged his acquaintance of things and people. Valuable and interesting stores of information about the products, traditions and legends of this old province of Poitou have been bequeathed to posterity in his works.

From the time that Rabelais entered upon his duties as secretary to the Bishop until September 16, 1530, when he matriculated as a medical student in the University of Montpellier, six years elapsed. During the last three, we find him sojourning for variable periods at various universities. During this period he finally casts aside his Benedictine garb and is dressed in the habit of a secular priest, entailing the breaking of his vows as a monk and incurring ecclesiastical censure for the crime of apostacy. Sometime afterward, as we shall see, he obtained without much difficulty absolution.

The method of getting higher education by wandering from one university to another and attending the courses in which each specialized, was common practice in Europe at that time and such a method has much to commend it for consideration even now.

While in the service of Bishop d'Estissac, Rabelais found opportunities to attend lectures of the Legal Faculty in the neighboring city of Poitiers. In his three years of pilgrimage, Rabelais visited the universities of Bordeaux, Toulouse, Bourges, Orleans and finally Paris. No record is found of his matriculation as a medical student in any of these universities, but he must have pursued some medical studies before entering the Medical School of Montpellier. For, six weeks after admission, he emerged with his degree of Bachelor of Medicine. Apparently it was merely a question of passing the required examinations, for the period of study required for this degree was ordinarily not less than two years. To attain the Master's degree the statutes prescribed six years of study. This Rabelais did not obtain until 1537.

The Medical School of the University of Montpellier, founded early in the thirteenth century, had acquired a great reputation throughout Europe and at that time was second only in prestige to the Medical School at Padua. After receiving his degree, Rabelais was appointed lecturer and in 1531 gave a series of lectures at the University on anatomy and upon the Greek masters of medicine, Hippocrates and Galen, which in many ways were a learned curiosity, for he commented upon them directly from the Greek text.

There is evidence that, as a University student, Rabelais was not the pedant and bookworm that might be imagined from his erudition. He indulged in the sports and secular activities of college life and at Montpellier took part in the performance of farces and is credited with writing a comedy, "The Man Who Married a Dumb Wife."

In the Spring of 1532, at the age of thirty-eight, Ravelais moved to Lyons, at

that time a center of commercial and intellectual activity, the headquarters of an unusually enlightened society of which he became a part. Here, in particular, the new printing and publishing industry was centered. For some years Lyons was the site of Rabelais' medical and literary activities. In the very first year, from the Greek text, he translated, edited and annotated for publication by a Lyonaise printer, the medical works of Hippocrates and Galen. The importance of this service to the medical thought and practice of today will be shown later.

The publication of these works probably helped secure for the author his appointment on November 1, 1532 as physician to the Hôtel Dieu, probably the largest and oldest hospital in France at that time. Legend attributes the founding of this hospital to the sixth century. It was devoted entirely to the care of the sick poor. It had an ample endowment, was well appointed, and had an adequate staff of nurses and attendants. It accommodated about two hundred patients. We could hardly call it a two hundred bed hospital, for at that time their large beds accommodated two and sometimes three patients. Here the barber-surgeon accompanied Rabelais on his rounds and executed operations under his supervision. The salary was about $200 a year. No doubt a desire to add to his income was an additional incentive for writing, as well as Rabelais' natural urge toward literature.

Composed in leisure moments, during his first year of medical practice, he published in 1535 "The Horrible and Dreadful Feats and Prowesses of the Most Renowned Pantagruel." In the complete work this forms the second book. The widespread popularity and success that followed encouraged Rabelais to follow it up with "Gargantua," which now forms the first book. Later, the story was continued in three other installments.

In this period, too, Rabelais began the publication of a series of humerous almanacks, "Pantagrueline Prognostications," that may have served as an inspiration to Benjamin Franklin. The fame of Pantagruel soon reached Paris, where the book became the talk of the town and where it received the censure of the Sorbonne.

From time to time Rabelais revisited and lectured at Montpellier. At Lyons he renewed friendship with Jean du Balley, one of the brothers mentioned as early school friends, who had now become Bishop of Paris, with great prestige at Court by reason of his cleverness in diplomacy. In the year 1535 the Bishop was sent by King Francis on a diplomatic mission to Rome, that involved intercession with the Pope to withold his excommunication of Henry VIII of England. The Bishop engaged Rabelais to accompany him as physician. On this, the first of three visits to Rome, he stayed two months.

Rabelais allowed his hospital connection at the Hôtel Dieu to lapse after a service of three years. Old records have preserved the information that the death rate decreased between 2 and 3 per cent during his incumbency. We soon find him again accompanying Jean du Balley, now a Cardinal, to Rome. He resided there this time for about seven months.

The Pope at that time was Paul III, Alesandro Farnese, rich scion of Roman nobility, patron of art and letters. Rabelais with the backing of his influential Cardinal friend, requested from the Pope absolution for having laid aside the Benedictine garb without authorization. In January, 1536 the Pope granted him an indult, which authorized him to return to a Benedictine convent, at his choice, and to practice medicine provided that, according to the customary formula, he used neither cautery nor the knife and intended to make no profit thereby.

On his return from Rome, Rabelais spent a few months at a Benedictine monastery near Paris, where his friend, the Cardinal, secured for him the title and stipend of canon. But we find him soon again engaged in medical practice, which was to be his career for the next ten years. He first went

to Montpellier, where in May, 1537, he was awarded with due formality his degree of Master of Medicine. The summer of the same year saw him again practicing in Lyons and it is recorded that, before an assembly of savants, he gave a public dissection and lecture on human anatomy, an event worthy of note at that time in France, when dissection was rare even in medical schools.

In the Fall he delivered a term's course of lectures at Montpellier. After practicing for two years in Lyons, Rabelais in 1539 entered the service of Guillaume du Balley, elder brother of the Cardinal, and accompanied him as physician and member of his staff to Turin, where this eminent soldier ruled Piedmont as Governor.

Rabelais remained in this service for nearly four years until the death of the Governor. While at Turin he had published an edition of "Gargantua and Pantagruel" together (both had been repeatedly reprinted separately) in which some too pungent expressions were modified.

Of the two years of Rabelais' life following his return from Turin nothing at all is known as to his whereabouts or occupation; but in 1546 the Third Book of Pantagruel appeared "avec privilege du Roi," which had been given him in September, 1545. We know that he had been honored by King Francis with the title of Master of the King's Requests, a title bestowed upon distinguished men of letters, which accounts for the King's endorsement of his new book.

While there is no positive evidence of any measures taken or threatened against Rabelais, it is true, nevertheless, that during this two-year period of his life a storm of persecution was gathering force, directed at first against Lutherans and claiming now, as victims, some of the Humanists. It is probable that Rabelais in consequence stepped out of public notice and for that reason no record remains.

In spite of having obtained from the King "privilege to print" and in spite of greater prudence in his new work, the Third Book was condemned by the Sorbonne as the first two had been. This action annoyed Rabelais and in March, 1546, he left France, crossed the frontier to Metz in Loraine, where he accepted a position as city physician at about three times the salary he had received at Lyons and resided there about two years. While there he began the Fourth Book of his masterpiece and issued an almanac for the year 1548.

Francis I died in 1547 and was succeeded by Henry II, who sent Cardinal du Balley as ambassador to Rome, He needed a doctor, so Rabelais made with him another and last visit to Rome, which occupied the greater part of the year 1548.

In 1550, through the influence of his friend, the Cardinal, he was appointed curé of Meudon and received also another ecclesiastical benefice. He received the income from these during the last three years of his life, but it seems quite certain that he did no duty as curé or as priest. During these years he seems to have lived chiefly at the palace of Cardinal du Balley in the outskirts of Paris. Here he finished in 1551 the fourth of his Pantagruelian Mythologies. For the printing King Henry II grants "his royal privilege and particular protection for the author against his slandering adversaries." At this time relations were strained between the French King and the Pope. Rabelais, in this Fourth Book, shows his appreciation of Royal favor by concentrating his satire upon the temporal ambitions of the Holy See. Pope Julius III offered rather a good mark, for he was notoriously worldly in his tastes and his administration noted for its nepotism. The Sorbonne had supported the King against the Pope, but by the time the book was published, the differences had been ironed out and the book was banned by the Sorbonne.

How Rabelais spent the last year of his life following this condemnation is not definitely known. He died in Paris in August of 1553 and was buried in St. Paul's cemetery, according to one account in the

nave of the church. Some of the legends about his death would no doubt have pleased Rabelais himself. One is that his last words were "Draw the curtain: the farce is ended"; another that his last will was in the form of a jest: "I possess nothing; the rest I give to the poor." There is some doubt as to the authenticity of a Fifth Book, continuing the story of Pantagruel, which is included in editions of his works. It first appeared in 1562, nearly ten years after his death.

Four centuries have passed since the first installment of "Gargantua and Pantagruel" appeared. During that time few books have been more often reprinted. The style has had thousands of imitators. To pick a few at random, we see the influence in the writings of Sterne, Swift, Molière and Voltaire as well as perhaps Bernard Shaw and James Joyce.

Appraisal of the author of this work ranges from the creator of French literature and the greatest of philosophers to the "infamous Rabelais" and "the mud-heap of humanity." There is nearly unanimity of opinion among literary critics, however, that Rabelais did for the French, what his contemporary Cervantes did for Spanish literature. Breaking away from the Latin of scholasticism and expressing his ideas in the vulgar tongue, Rabelais is ranked as a pioneer in the establishment of French prose literature in its modern form.

The story of "Gargantua and Pantagruel" is ostensibly a fairy story about a family of giants, Grandgousier, his son, Gargantua, and his grandson, Pantagruel, who as a youth, amused himself one day by plucking the bells from the tower of Notre Dame to hang on his mare's neck. They are enormous fellows, great drinkers, fighters and disputants. Nearly all agree that, as a story teller, Rabelais was endowed with a wealth of imagination that would qualify him as a charter member of the "Tall Story Club."

Agreement is general, too, that some passages are very coarse and obscene and some, according to our present canons of good taste, almost unreadable at the present day. As a result, a modern conception has been built up from reading him in prurient extracts. The form of the book is essentially popular. If compared with contemporaneous farces and comedies, its indecency is not so marked. At any rate his coarseness is not marred by the type of subtle voluptuousness that we tolerate in much modern writing. These greatly exaggerated obscene delineations of vulgarity and vice do not form the main theme and are counterbalanced by many almost Utopian ideals of virtue.

During his long sojourn in cloister and university, Rabelais had acquired a vast store of learning, Greek and Latin literature, philosophy, theology, law and medicine. Into this story Rabelais, endowed also with a remarkable trait of good-humored satire, interweaves a commentary on the customs, the thoughts, feelings, aspirations and knowledge of that critical period in European history. His mind is permeated with the aspirations of the Humanists for social, political religious and educational improvement and above all for a joyful, non-ascetic life. These ideas permeated this extremely humerous book, making fun of all the follies of his day, combining burlesque, allegory, extravaganza, and covert satire into a distinctive type of humor, that bears the name of Pantagruelism.

Education lagged behind the discoveries and needs of the age in the sixteenth century even more than it does in our time. It had become crystalized in the traditional study of ponderous Latin treatises and the mysteries of Scholastic Philosophy. Rabelais' dislike and antipathy toward what seemed an antiquated and inadequate program of education finds utterance in "Gargantua and Pantagruel," where, in a satire on the school system of the Middle Ages, he contrasts the early education of Gargantua with all the best and noblest ideas of the Humanist Renaissance in reference to pedagogy. The practical application is embodied in the description of an ideal co-educational Abbey, Theléme,

which Gargantua founds and endows. Here the curriculum includes science, history, music, painting, trade practices as well as sports and a gymnasium.

The Humanist reformers aspired, also, to regenerate the legal practice of France with a return to the old Roman jurisprudence as their ideal. Due to the background of Rabelais' study of law and association with eminent lawyers, his works abound in unexpected touches of legal erudition and the jargon of the law courts. He satirizes the undue delays, miscarriages, corruption and bribery in the administration of justice. A burlesque trial over which Pantagruel presides deftly presents the contrast between the ideals of Humanism and the medieval traditions that hampered the teaching and practice of law.

War, its follies, its tactics and technique, is not spared the shafts of his satiric ridicule, when he describes the Great War between Gargantua and Picrochele, threatening the involvement of the whole world all about a few stolen cakes. Gargantua's speech to the vanquished and his terms to the vanquished and his terms for reconciliation give an ideal of clemency and chivalry that by innuendo satirizes the actual treatment of conquered peoples by Christian nations.

Humanism had directed itself to the re-study of Biblical writ and became insistent upon re-establishing the Church on the basis of the original Gospels, open freely to study by all in the common language. It would free Christianity of the accretions of superstition that had attached to it during the centuries and from the spiritual despotism that had developed around the Papacy with many accompanying abuses. "It became a widespread revolt against the scholarship of the Middle Ages, that had turned the ancient Hebrew songs and histories and folk-lore, and even the simple messages of Jesus, into formidable parodies by expoundings, interpretations and allegories and so created a terrifying theology instead of a religion."

In his book Rabelais is in harmony with this viewpoint. He holds up to ridicule the habits, the indolence, the ignorance and gluttony of the monks. He travesties the pedantry and scholasticism of the Clergy. In his peculiarly daring and humorous way, Rabelais sets forth his liberal ideas about the accretions engrafted on Christianity, that seem to him unessential and misleading. Among them he includes the cult of the Virgin and of the Saints, the veneration of relics, pilgrimages and indulgences. He even satirizes the worldliness, pretensions and ambition of the Papacy of the time. His daring in this respect, however, did not exceed that of many other Catholic writers, whose orthodoxy is above suspicion. His satirical criticism is kindly compared with the bitter polemics of Luther and no more daring in their defiance of the proprieties.

On the other hand Rabelais had no sympathy for Protestantism. The dogmatic theocracy developed by Calvin was as much anathema to him as the theological defenders of orthodoxy at the Sorbonne. Like Erasmus he was against intolerance wherever he saw it. The zealots from each of these camps gave him the reputation of an atheist. His writings give evidence that he regarded the atheist as the most degraded type of all.

Time works its changes. Progress goes on then and now according to its own laws, usually in spite of, sometimes assisted and accelerated by the efforts of Humanists and Reformers. In the Catholic Church the monastic system was re-organized, the pretensions of the Vatican to civil and religious despotism modified and spirituality replaced worldliness and political ambition in the Papacy. On the Protestant side the dogmatism and intolerance has been modified.

Did the writings of Rabelais aid in bringing about this change or, as his detractors assert, was his influence entirely anti-religious and anti-spiritual? At any rate he remained a Catholic during his entire life and was a good Catholic perhaps in about the same sense that Robert La

Follette was a good Republican all his life. That depends upon the viewpoint.

It may be easier to evaluate Rabelais as a physician. If he was heretical, it speaks well for his professional skill, that for twenty years he was personal physician to one of the most eminent cardinals in the Church.

Rabelais' "Gargantua and Pantagruel" is everywhere enlivened by humorous medical quips and grotesque situations, that only a doctor with a keen sense of humor could imagine and only a doctor with some Pantagruelism in his make-up would wholly appreciate. If you seek merely diversion in reading him, I recommend for perusal Chapter VI, Book I, entitled "How Gargantua was Born in a Strange Manner." At a great outdoor feast in a willow grove Gargantua was born "upon the grass under the willows. The event did not interrupt the festivities and since he was born by way of his mother's left ear, there seemed no great impropriety in the accouchment being carried on more or less in public."

If you are uncertain as to the meaning of Pantagruelism, read the last paragraph.

> I doubt me that you do not thoroughly believe the truth of this strange nativity. Though you believe it not, I care not much. But an honest man and of good judgement believeth still what is told him and that which he finds written. Is this beyond our law or our faith? Against reason or the Holy Scriptures? For my part I find nothing in the Sacred Bible that is against it; but tell me if it had been the will of God, would you say he could not do it? Grammercy; I beseech you, never dumbfound your heads with these idle conceits; for I tell you, nothing is impossible with God; and if he pleased, all women henceforth should bring forth their children from the ear.

Was there ever better parody of some of the specious reasoning about miracles and authority?

But there is a serious importance and significance attached to the writings of Rabelais, which has great bearing upon the development of medicine, and it is this influence that I should like to make clear. The key to it was his interest in the original Greek texts of the classical masters of medicine. The original medicine man was also a priest and ever since it has been extremely difficult, for any length of time, to keep medicine free from religious and mystic superstition.

Hippocrates founded a Greek School of Medicine, emancipated from religion, and based on a true scientific spirit of observation and study of the nature of health and disease. By some freak of fortune his writings have been preserved for 2300 years and at opportune moments have come to light to direct medical thought out of the darkness of mysticism and superstition.

The Alexandrian School continued the concept of the Greeks about medicine and added to it the practical investigation of human anatomy and vivisection of animals. Then followed centuries in which these fundamentals were obscured by philosophic and mystic theories, until in the second century Galen, another Greek, from Pergamum, re-established the scientific method of his ancestors and of the Alexandrians and added much to anatomical and physiological knowledge as well as that of diagnosis and treatment.

Then, after the Fall of Rome, nearly all this was forgotten until near the end of the Middle Ages. But the texts of Hippocrates, Aristotle and Galen found their way to the Mohammedan scholars of Persia and Arabia and there served to build up another fine school of physicians, the Arabian School.

Eventually among them, however, the teachings of Hippocrates and Galen became overlaid with philosophy, alchemy and mysticism and their teaching stereotyped, authoritative, and lacking in research and original thinking. In this modified and much translated form, the teachings of Galen were again returned to Western Europe by the Arabians and the point from which they spread was the School of Salerno in Italy.

Then, unfortunately, the writings of Galen, instead of serving as a stimulus for research and original thought, became for centuries an authority for physicians as rigorous as the Bible for the Clergy. The Church, too, regarded with suspicion anyone who endeavored to explain disease on a natural basis or to investigate human anatomy or physiology. Alchemy and astrology also permeated and obscured the medical sciences. Quackery and charlatanism thrived all over Europe.

Strange as it may appear, it was this Catholic priest, Rabelais, the Curé of Meudon, who translated the original Greek texts of Hippocrates and Galen, annotated them and gave to the profession their true meaning and significance. Rabelais was of that small but scholarly, distinguished and influential group of medical Humanists of which Thomas Linacre in England was another eminent example. Theirs was the influence that started the re-birth of medicine on a scientific basis.

This new attitude and approach to medical problems culminated in the following century by producing, in Italy, that outstanding anatomist, Vesalius; in France, the great surgeon, Paré, and in England Harvey, immortalized by his discovery of the circulation of the blood.

Besides crediting Rabelais with a share in this influence toward re-directing medical thought, we also recognize in him a clever anatomist, an inspiring teacher and lecturer; one who, by the wit and satire of his writings, served the profession by unmasking the ignorance of the charlatan and asserting the independence of the physician from clerical restrictions.

The theologians and moralists may wrangle and argue about his character and writings. The medical profession gratefully recognizes François Rabelais as a medical leader of the sixteenth century.

BOOK REVIEWS

THE TREATMENT OF FRACTURES. By Dr. Lorenz Böhler. 4th English Ed., Trans. from the 4th Enlarged and Revised German Ed. By Ernest W. Hey Groves, M.D. Balt., William Wood and Co., 1935.

This is a well-reproduced translation of the fourth German edition of Böhler's book. It is good to have this important work available in English.

New material on the treatment of fractures of the calcaneus and of the cervical spine has been added to the English edition which may now be considered as the last word to date on this subject.

As Dr. Groves so well points out, no one has done more than Dr. Böhler "to demonstrate the importance of proper treatment of fractures, the necessity for unity of control, loyal and efficient team work, accurate knowledge of the after-results, and meticulous attention to detail." Whether agreeing with all of Dr. Böhler's methods or not, everyone treating fractures will want to and, in fact should, study this work thoroughly.

SURGICAL PATHOLOGY OF THE PERITONEUM. By Arthur E. Hertzler, M.D. Phila., J. B. Lippincott Company, 1935.

This is the sixth volume of a series of ten monographs by Dr. Hertzler on surgical pathology. There are over 200 beautiful illustrations. The subject is completely covered and the author is too modest when he says: "As I review these pages I am impressed with the pitiable little I have to offer for these years of toil." Dr. Hertzler has succeeded in picking the wheat from the chaff and what is known of the pathology and physiology of the peritoneum will be found well digested in these pages. The author is a thorough student of the literature and adds to this an experience of forty years of keen observation. His book is well worth while.

APPARATUS AND TECHNIQUE FOR ROENTGENOGRAPHY OF THE CHEST. By Charles Weyl and S. Reid Warren, Jr. Springfield, Charles C. Thomas, 1935.

This book represents the last word in technique of chest roentgenography. The principles of physics are elaborated and apparatus and technique are carefully described. Not many roentgenologists would want to undertake all the minute details necessary in carrying out the work along the lines laid down by the authors. For the specialist, however, no better guide can be offered. The work must be considered as a classic in its own line.

A word must also be said for the splendid typography of this work. Special commendations are due to the illustrations which are unusually excellent and which are reproduced by the aquatone process which is too expensive for the average medical publication.

THE PRINCIPLES AND PRACTICE OF MEDICINE. By Sir William Osler. Ed. and rev. by Thomas McCrae, M.D. Ed. 12, Rev. N. Y., D. Appleton-Century Co., 1935.

Since the preparation of this book, word has come of the death of Dr. Thomas McCrae who has been revising Osler's famous work since Sir William's death. Under Dr. McCrae's editorship, the famous "Practice" of Sir William Osler has carried on and retained its vitality. With both the author and the reviser now dead, it is to be hoped that a new editor will be found who can continue this work which has been one of the landmarks of American Medical Literature.

As Dr. McCrae points out, there are changes and additions in practically every part of this book, which has been entirely reset. While larger than before, it has been kept within a convenient size by the selection of a new type page. The Osler tradition has been maintained in the emphasis on the clinical side of disease problems.

This book, representing the best traditions in medicine, may still be considered the outstanding work on clinical medicine.

THE KIDNEY IN HEALTH AND DISEASE. In Contributions by Eminent Authorities Ed. by Hilding Berglund, M.D. and Grace Medes, PH.D. With the Collaboration of G. Carl Huber, M.D., Warfield T. Longcope, M.D. and Alfred N. Richards, M.D. Phila., Lea & Febiger, 1935.

This is a well-edited and revised presentation of the papers read at the Symposium held in Minneapolis in the summer of 1930, on the subject of Kidney Disease, under the leadership of Dr. Hilding Berglund. There are forty-one contributions by the leading authorities and the subject is covered in a thorough manner that will make this a splendid work of reference. Such a work has been needed and will be found invaluable by those treating diseases of the kidney.

Thoracoplasty with Extrafascial Apicolysis. By Carl Semb. Suppl. to Vol. LXXVI, Acta Chirurgica Scandinavica.

A new method of obtaining complete collapse of the tuberculous upper lobe cavities is presented by Carl Semb of Oslo, in a beautifully illustrated and very well written monograph in English just issued as a supplement to Volume LXXVI of the Acta Chirurgica Scandinavica. The author feels that inadequate collapse is obtained in many upper stage thoracoplasties even though they are done by the modern method, which includes removal of the entire length of the first and second ribs. This is due to the lung being held upward and outward by its attachments along the spinal column, and to the great vessels and the brachial plexus at the top.

He feels that the method of apicolysis previously used to overcome this condition, namely, the separation of the lung in the line of cleavage between parietal pleura and periosteum and intercostal muscles, is not only difficult and dangerous, but frequently not efficient.

His method he calls extrafascial apicolysis as the separation is done outside the periosteum and intercostals. After complete removal of the upper ribs, all attachments of the lung plus the intercostal muscles and the periosteum are cut from the dome of the chest cavity. The scalene muscles are severed, all fibrous bands running to the brachial plexus and the subclavian artery and vein are divided, and then the periosteum of the first, second, and third ribs along with the corresponding intercostal muscle bundles and the intercostal nerves and vessels are completely divided close to the spine between ligatures.

The whole apex of the lung is thus freed, and is allowed to drop downward toward the hilum. When the periosteum slowly hardens directly on the lung later re-expansion is prevented.

Semb's results with this radical extrafascial apicolysis are remarkably good. In a series of 77 cases, he secured complete collapse of the cavity in more than 90 per cent; freedom from bacilli in over 80 per cent, with less than 3 per cent mortality. He operates under local anesthesia and usually removes as many as six ribs at one stage.

To most thoracic surgeons in this country, the removal of the upper six ribs plus the radical apicolysis will seem a dangerous procedure. The tendency since the introduction of the modern system of removing the entire length of the first and second ribs, has been to start with only the top three or possibly four ribs. The additional extensive freeing of lung by Semb's method certainly adds considerably to the time of the operation (Semb himself says it may add anywhere from ten minutes to an hour), to the shock and to the amount of tuberculinization produced by manipulation of the tissues. Semb's own statistics are, however, perfect arguments against these objections.

Since such excellent results are now being obtained in the clinics in this country with our present methods, it would certainly not seem advisable to adopt extrafascial apicolysis as a routine procedure. It does, however, appear to be a most valuable method, which can be called upon in those cases in which the cavity seems likely to retreat into the posterior gutter and remain only partially collapsed.

Modern Operative Surgery. Ed. 2. Ed. by G. Grey Turner, M.D. 2 vols. Balt., William Wood and Co., 1934.

First published in 1924, this is a thoroughly revised and up-to-date presentation of operative surgery by twenty-seven of the leading surgeons of England. Especially to be cited are the articles by Dr. Ernest W. Hey Groves on "Operations on Bones," Dr. W. Sampson Handley on "Principles of the Operative Treatment of Malignant Disease," and "Operations on the Breast," Dr. J. P. Lockhart-Mummery on "Operations for Stricture of the Rectum (Non-Malignant), Haemorrhoids, Fistula in Ano, Prolapse," Dr. L. Bathe Rawling on "Operations on the Skull and Brain," Sir Harold Gillies on "Plastic Surgery," Sir John Thomson-Walker on "Operations on the Kid-

ney and Ureter," "Operations on the Bladder," "Operations on the Prostate and Seminal Vesicles," and "Operations on the Urethra."

A Textbook of Surgery. By W. Wayne Babcock, M.D. Ed. 2. Phila. W. B. Saunders Co., 1935.

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Names of Surgical Operations. Ed. by Carl E. Black. St. Paul, Bruce Pub. Co., 1935.

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Parenthood: Design or Accident. By Michael Fielding, N. Y., Vangard, 1935.

Praktische Anatomie. By J. von Lanz and W. Wachsmuth. Berlin, Springer, 1935.

Puerperal Gynecology. By J. L. Bubis. Balt., Williams & Wilkins, 1935.

Rats, Lice and History. By Hans Zinsser. Boston, Little, Brown, 1935.

Réactions labyrinthique et équilibre. By G. G. J. Rademaker. Paris, Masson, 1935.

Romance of Exploration and Emergency First-aid from Stanley to Byrd (The). By Burroughs Wellcome and Co. N. Y., Burroughs Wellcome, 1934.

Synopsis of Regional Anatomy (A). By T. B. Johnston. Ed. 3, Phila., Lea & Febiger, 1935.

Theory and Practice of Anaesthesia. By M. D. Nosworthy. London, Hutchison Scient., 1935.

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